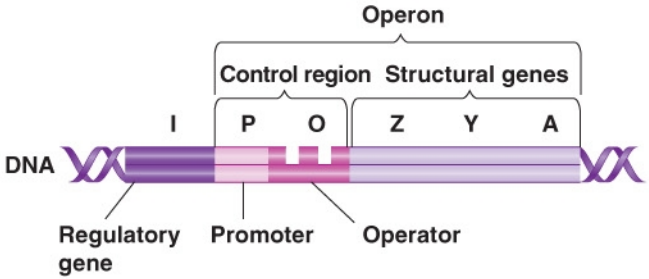
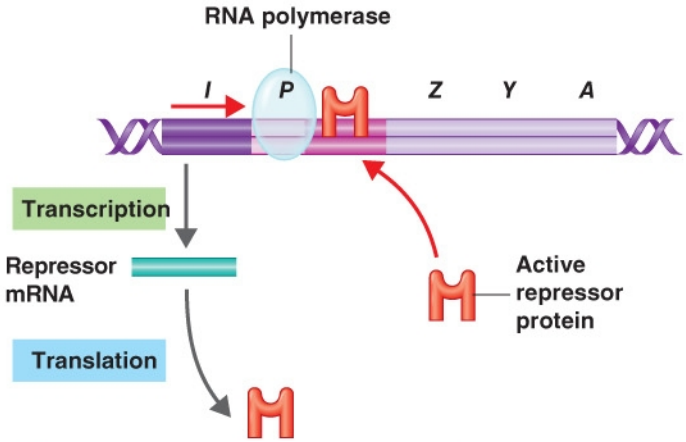
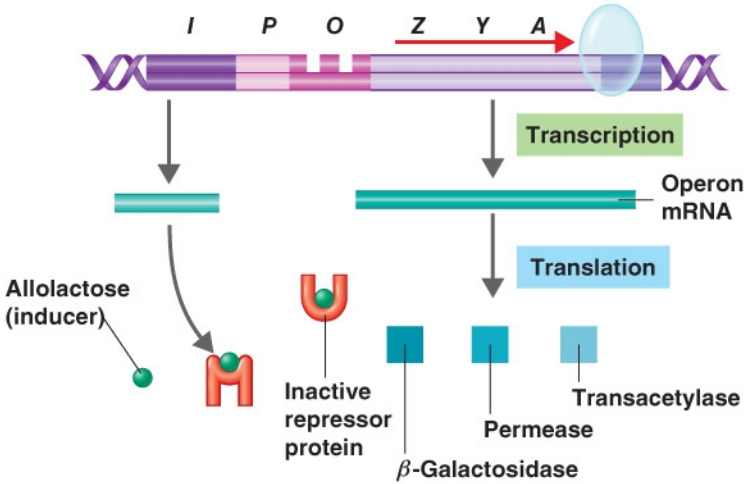
Operon model: Negative inducible



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*).



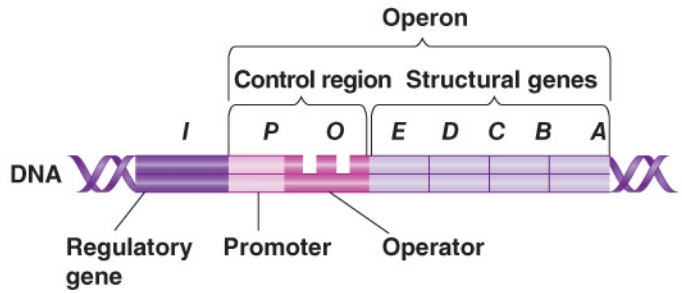
2 Repressor active, operon off. The repressor protein binds with the operator, preventing transcription from the operon.



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



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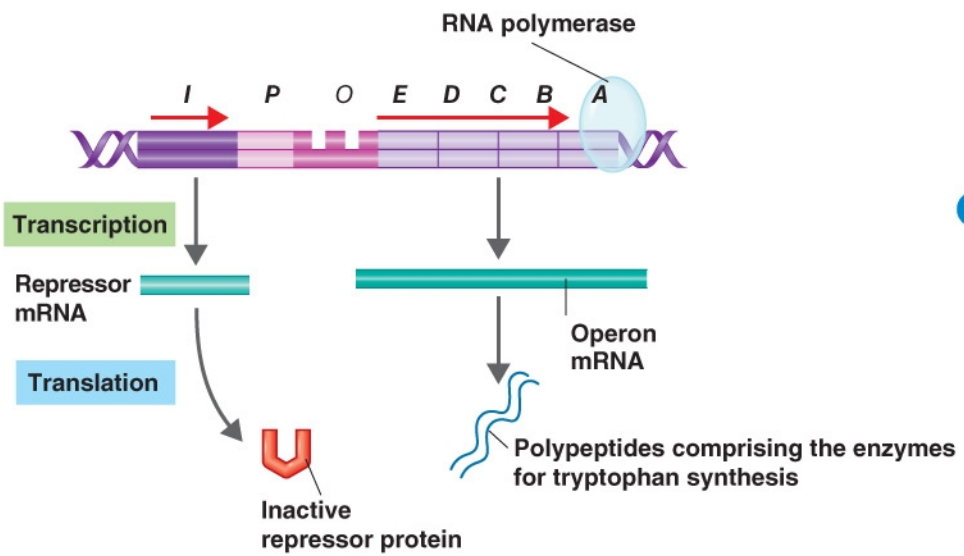
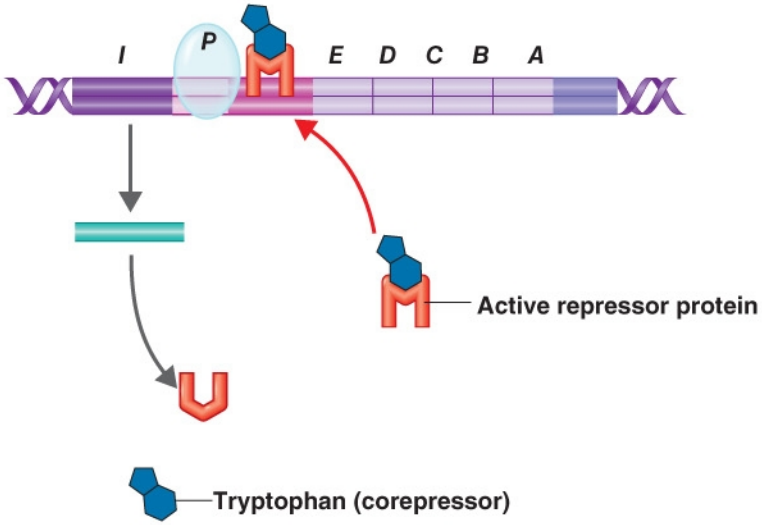


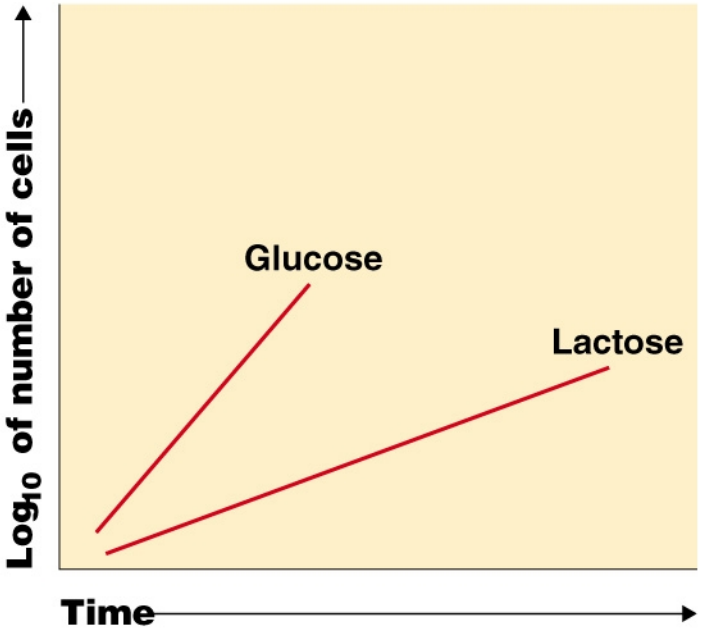
2 Repressor inactive, operon on. The repressor is inactive, and transcription and translation proceed, leading to the synthesis of tryptophan.



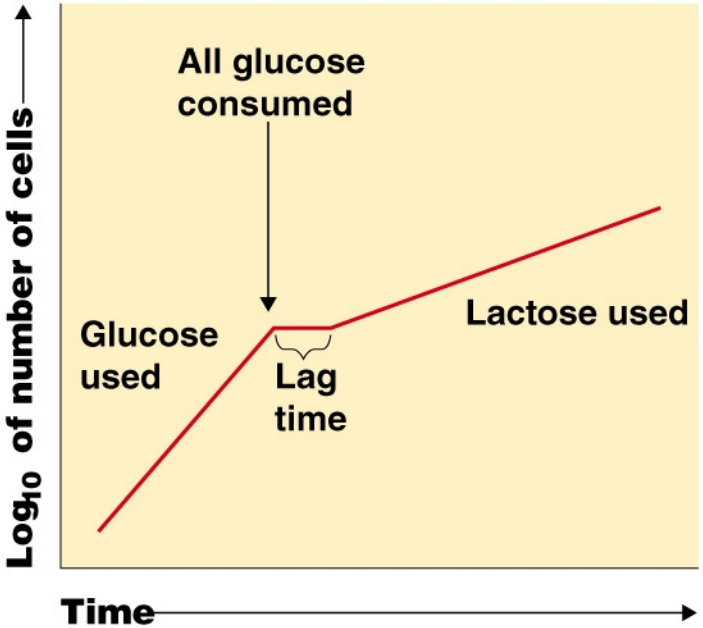
3 Repressor active, operon off. When the corepressor tryptophan binds to the repressor protein, the activated repressor binds with the operator, preventin transcription from the operon.

Fig. 8.13

Operon model



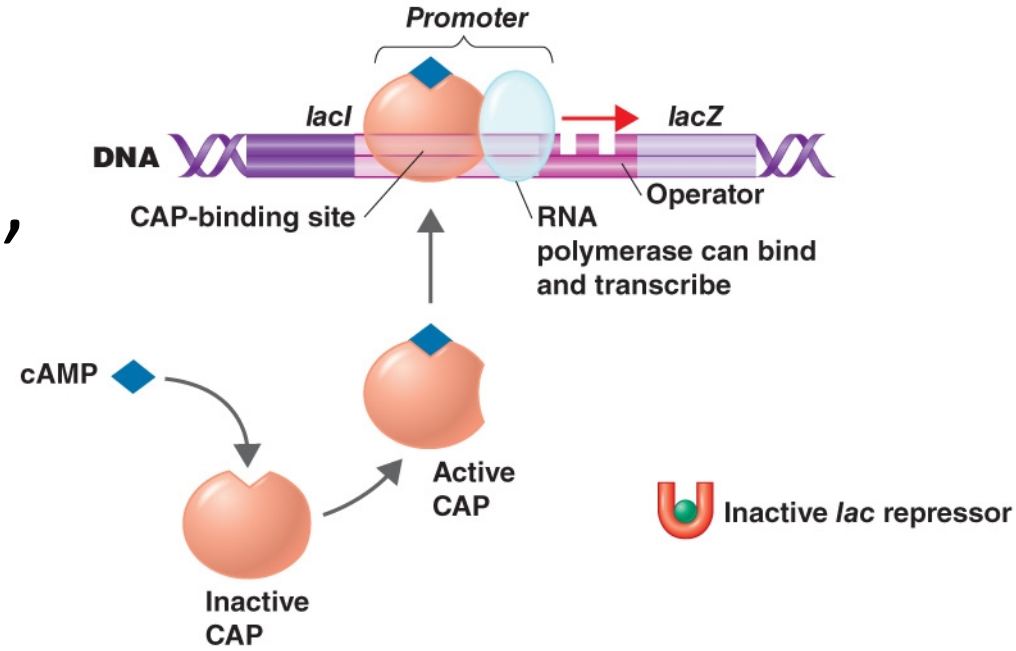
(a) Bacteria growing on glucose as the sole carbon source grow faster than on lactose.



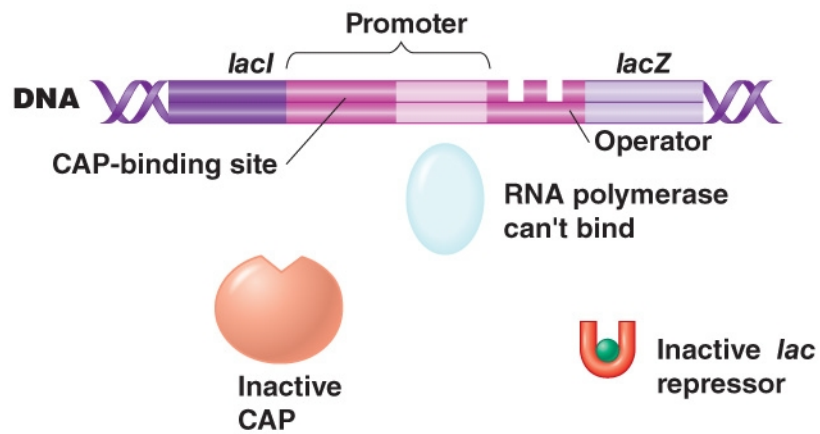
(b) Bacteria growing in a and lactose first consume the glucose and then, after a short lag time, the lactose. During the lag time, intra-cellular cAMP increases, the operon is transcribed, more lactose is transported into the cell, and β-galacto-sidase is synthesized to break down lactose.

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.

Fig. 8.15

Paper: Toledo-Arana *et al.* 2009

Mutations

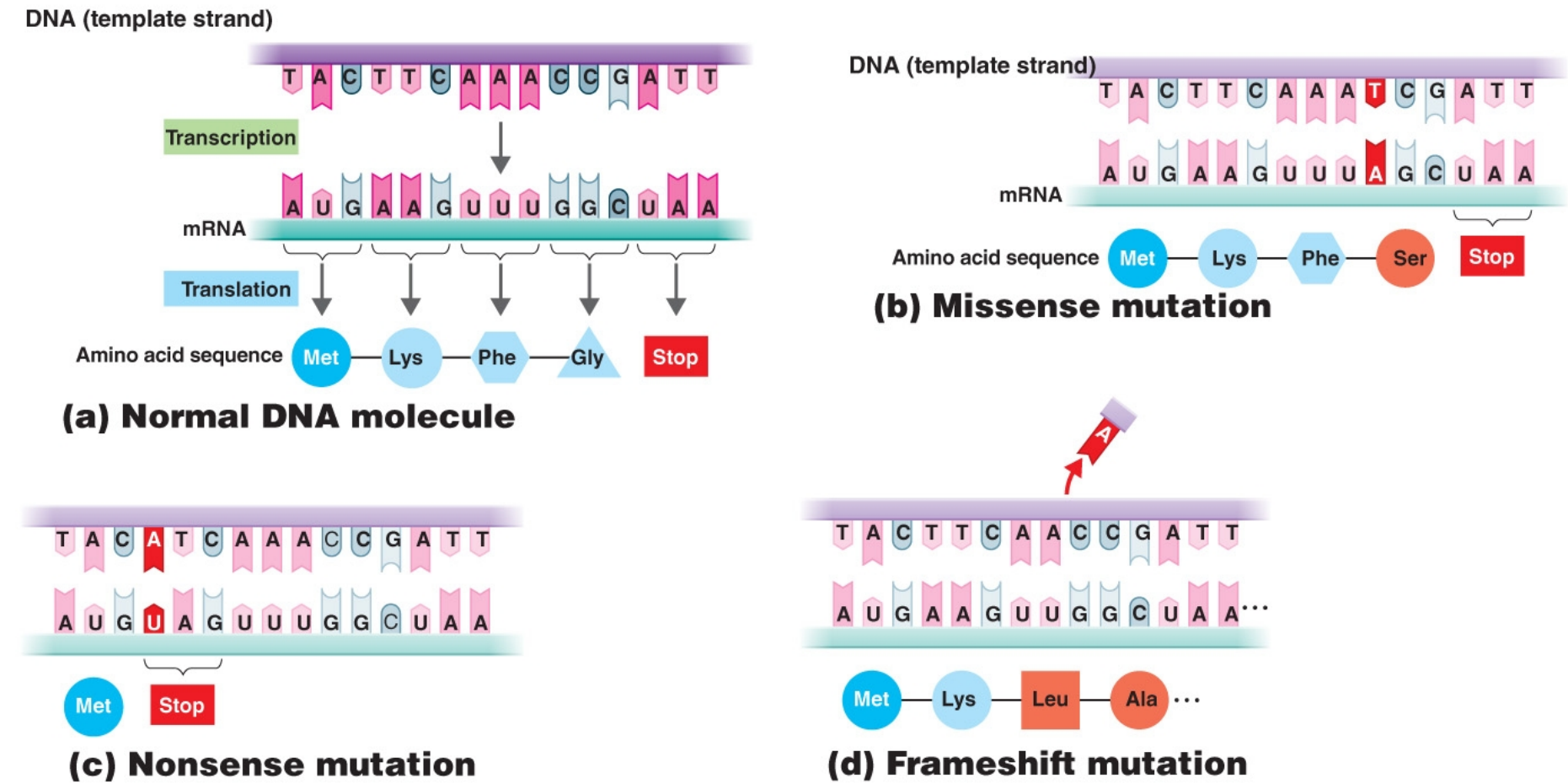


Fig. 8.18

Mutations

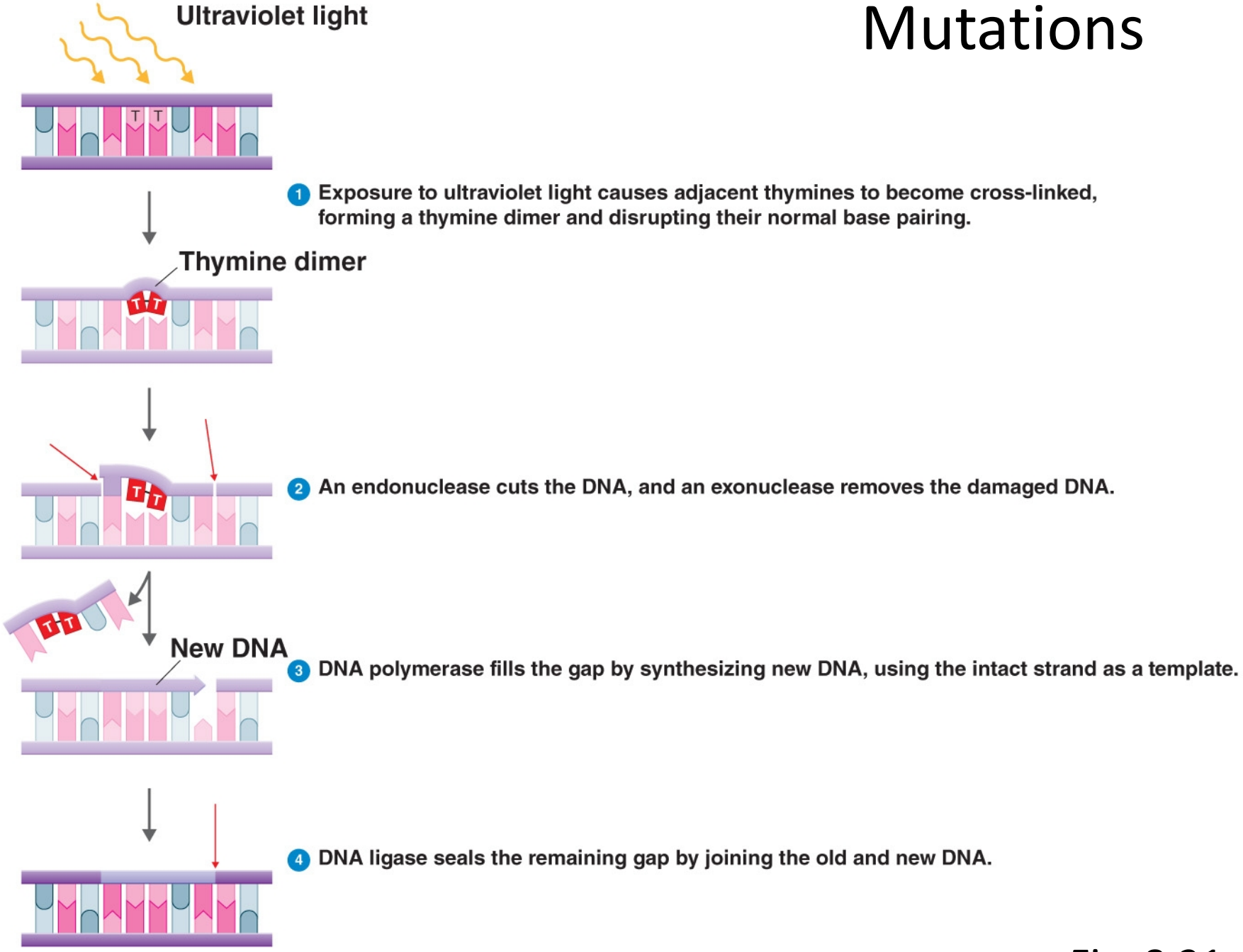
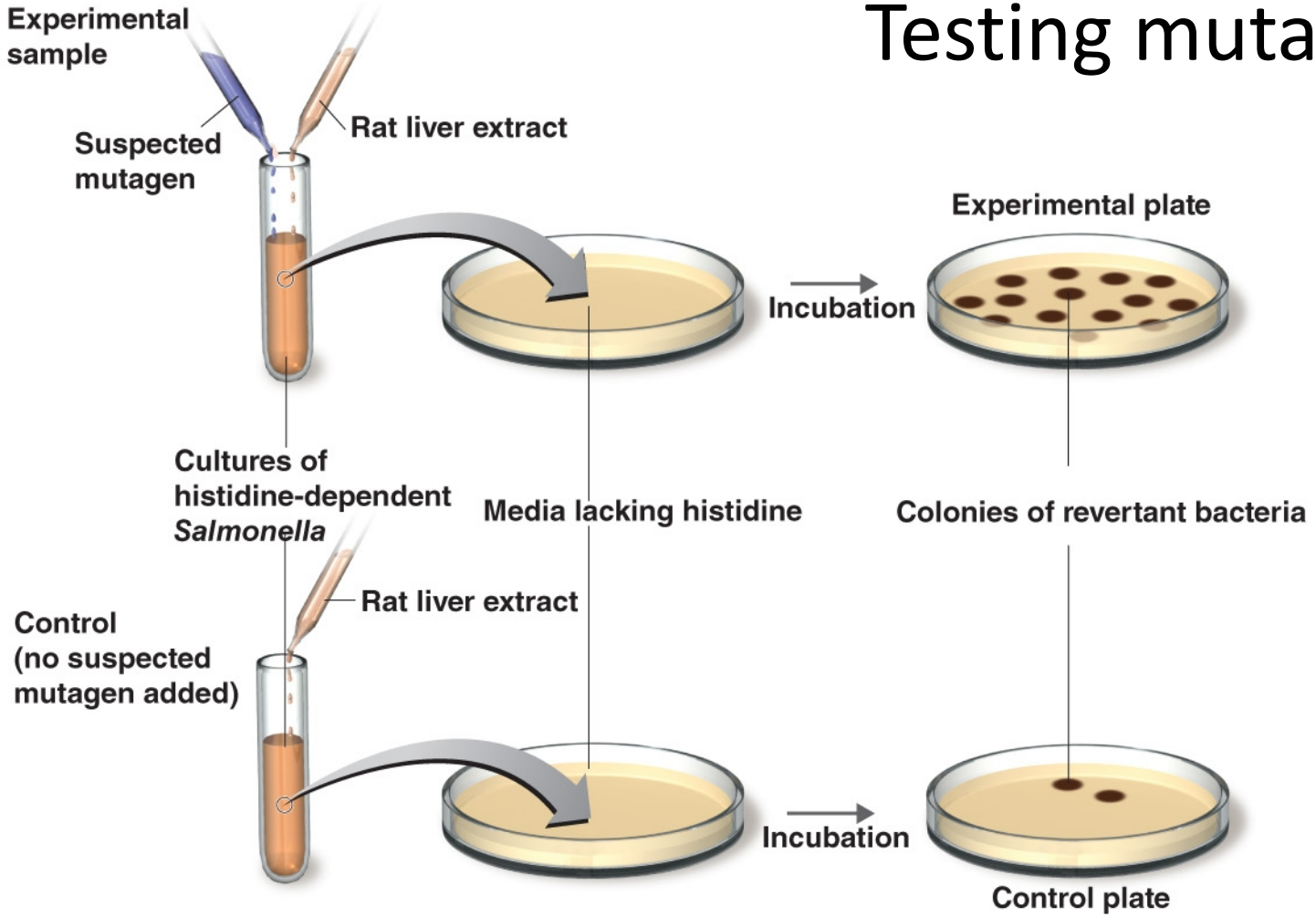


Fig. 8.21

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.
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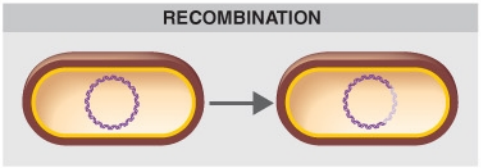
Fig. 8.23

Cyclophosphamide: a nitrogen mustard alkylating agent

en.wikipedia.org

A sign of recombination



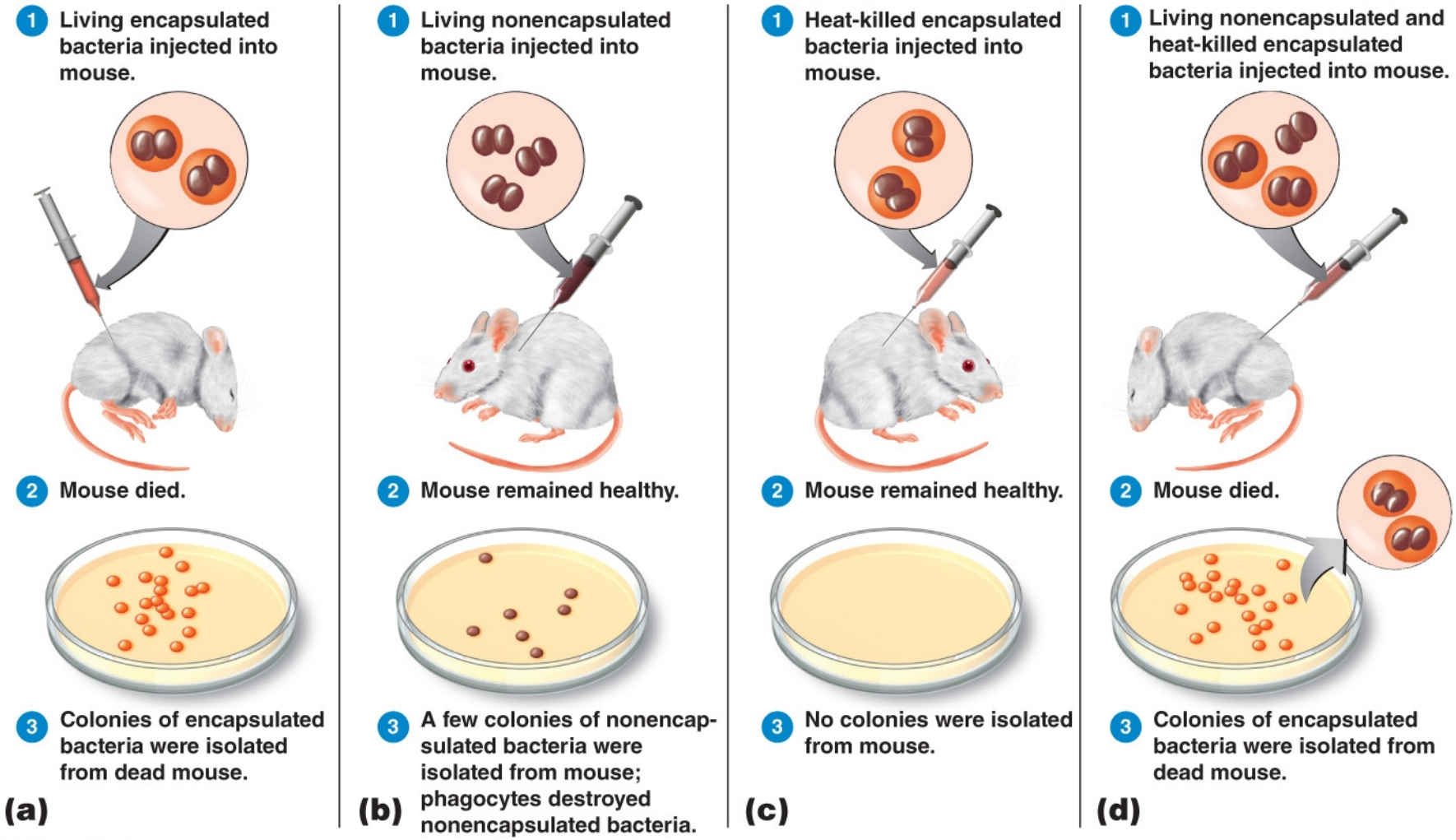


Fig. 8.25

Recombination: Transformation

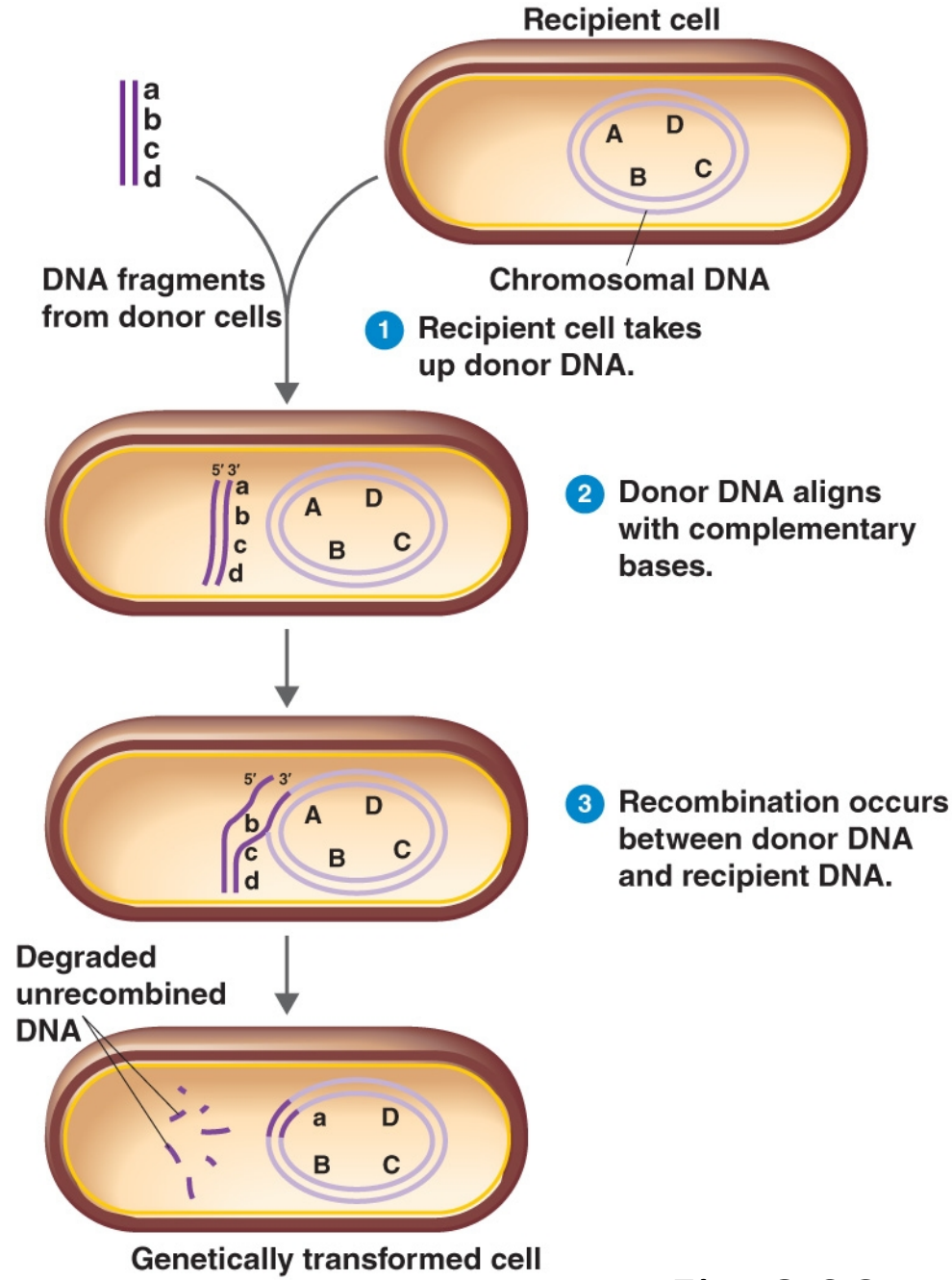
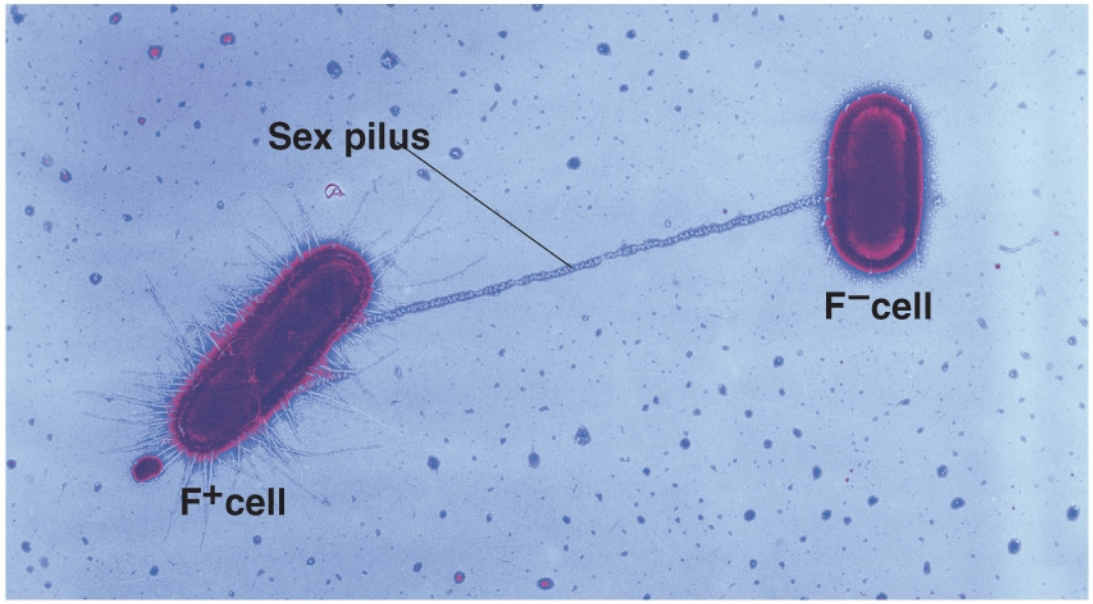


Fig. 8.26

Recombination: Conjugation



(a) Sex pilus

TEM 1 μm

(b) Mating bridge

TEM 0.3 μm

Fig. 8.27

Recombination: Transduction

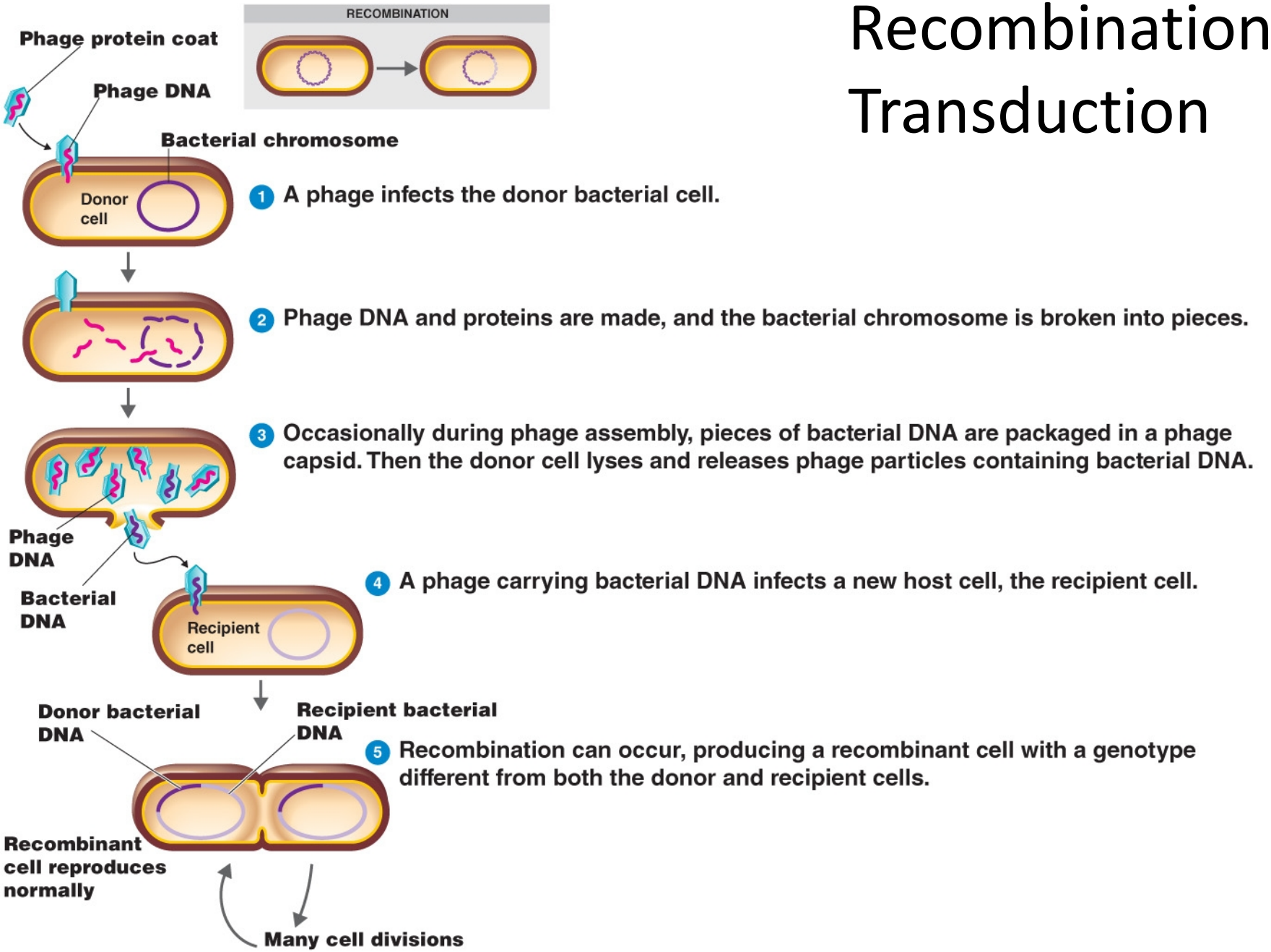


Fig. 8.29

Plasmids

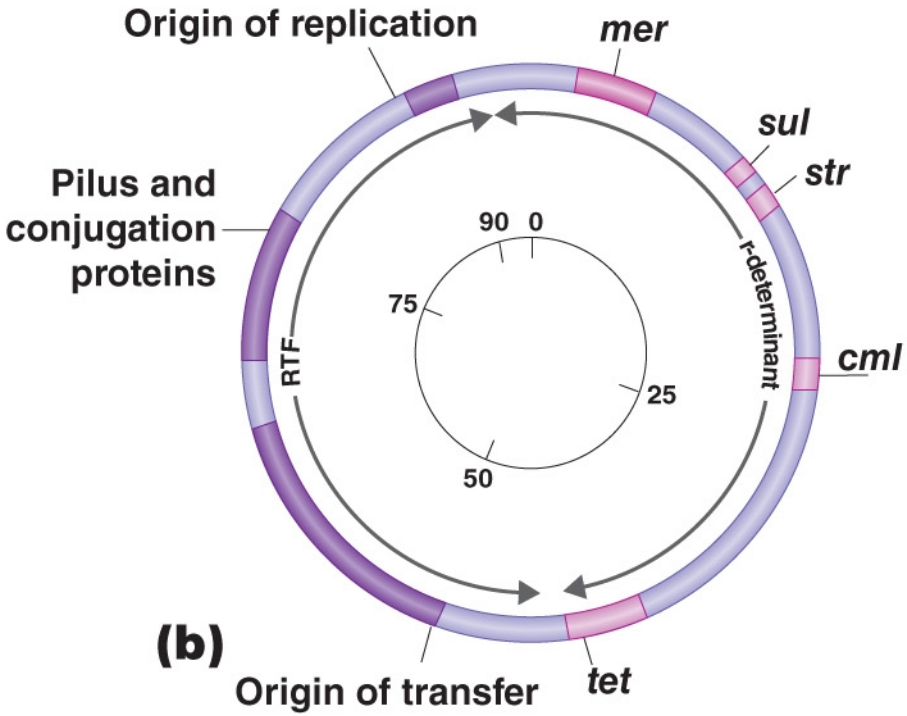


Fig. 8.30

Operon model

Fig. 8.13

Operon model

Fig. 8.13

Operon model: Negative inducible

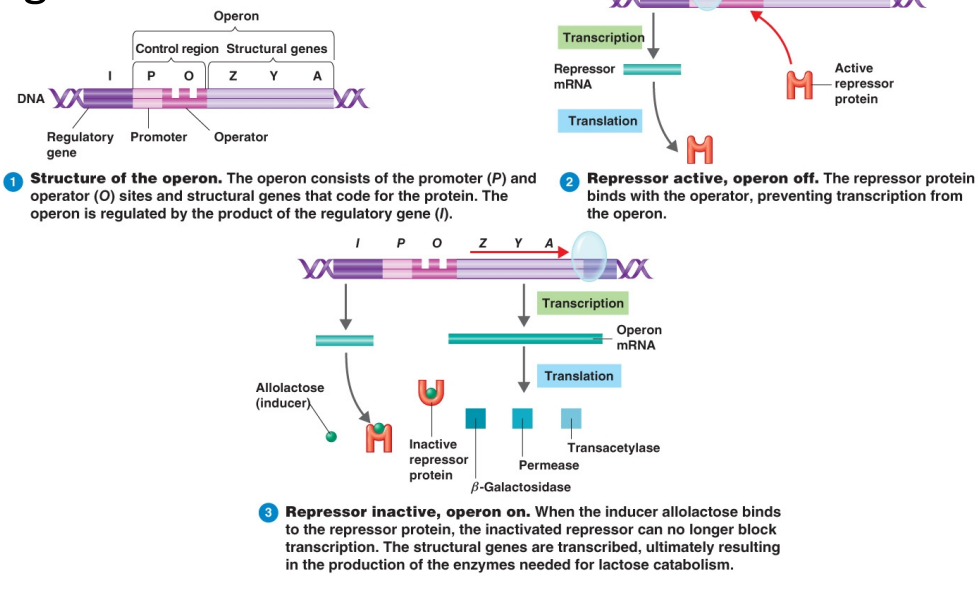


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Fig. 8.12

Operon model: negative repressible

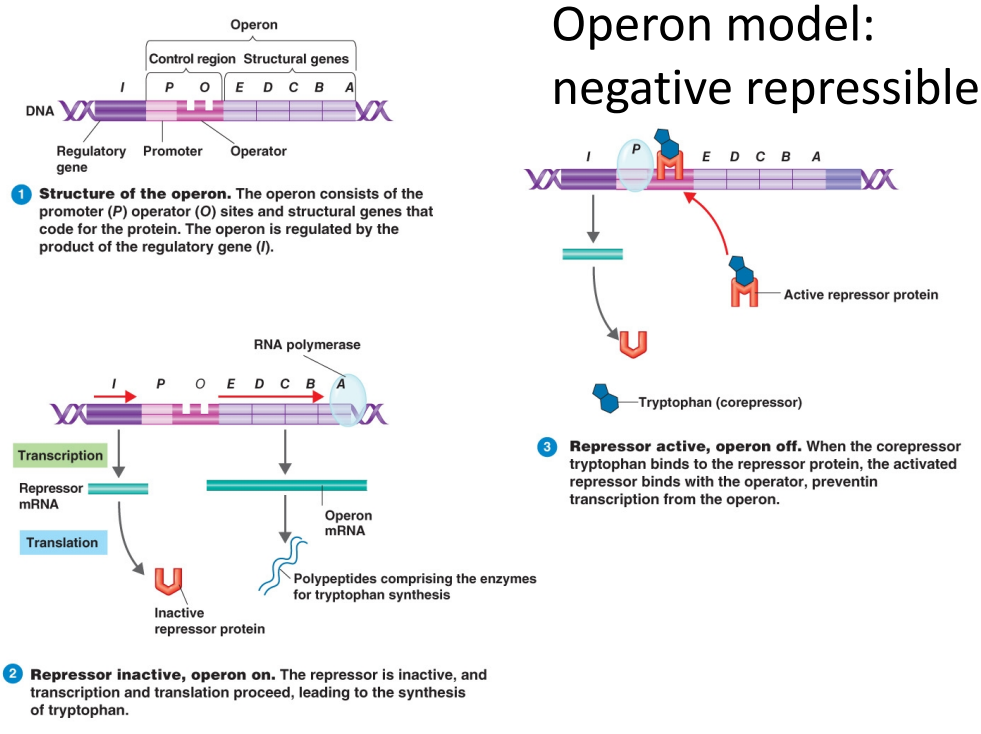

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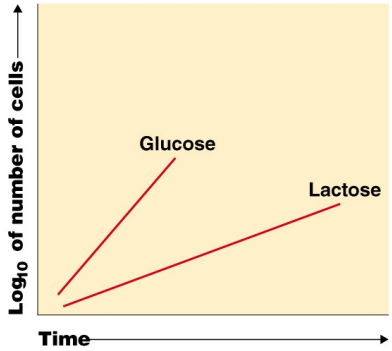
Fig. 8.13

Operon model



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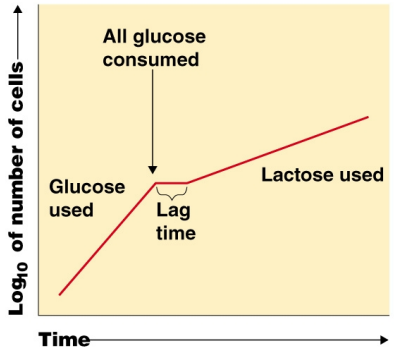

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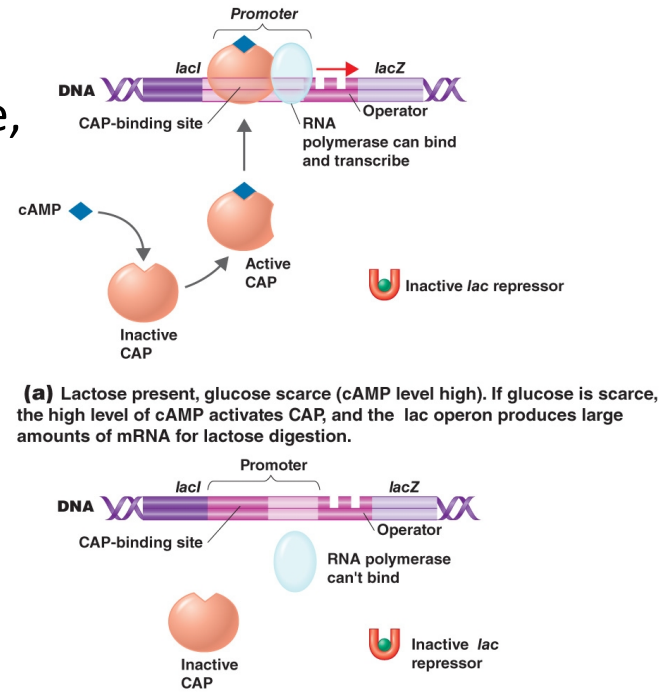

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Fig. 8.15

Paper: Toledo-Arana *et al.* 2009

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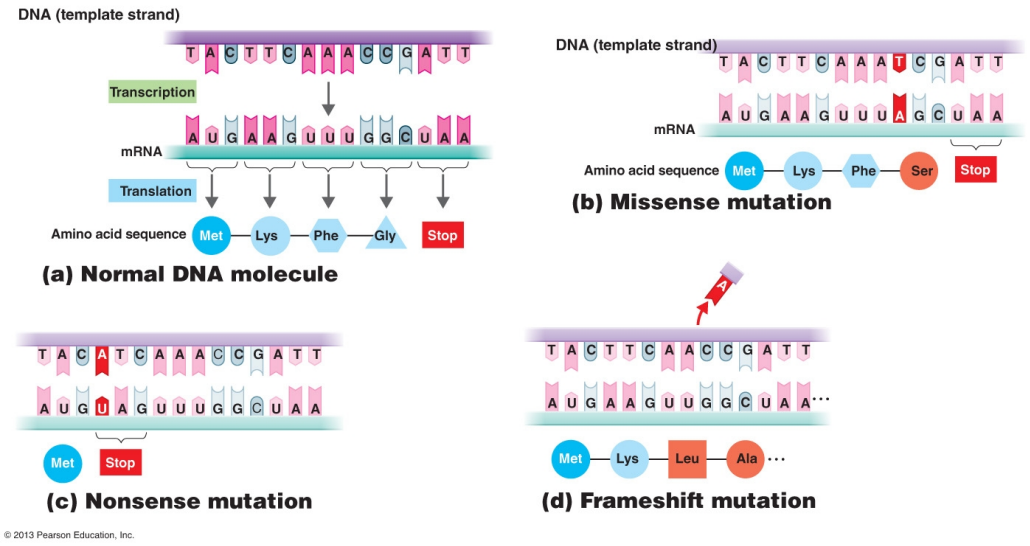


Fig. 8.18

Mutations

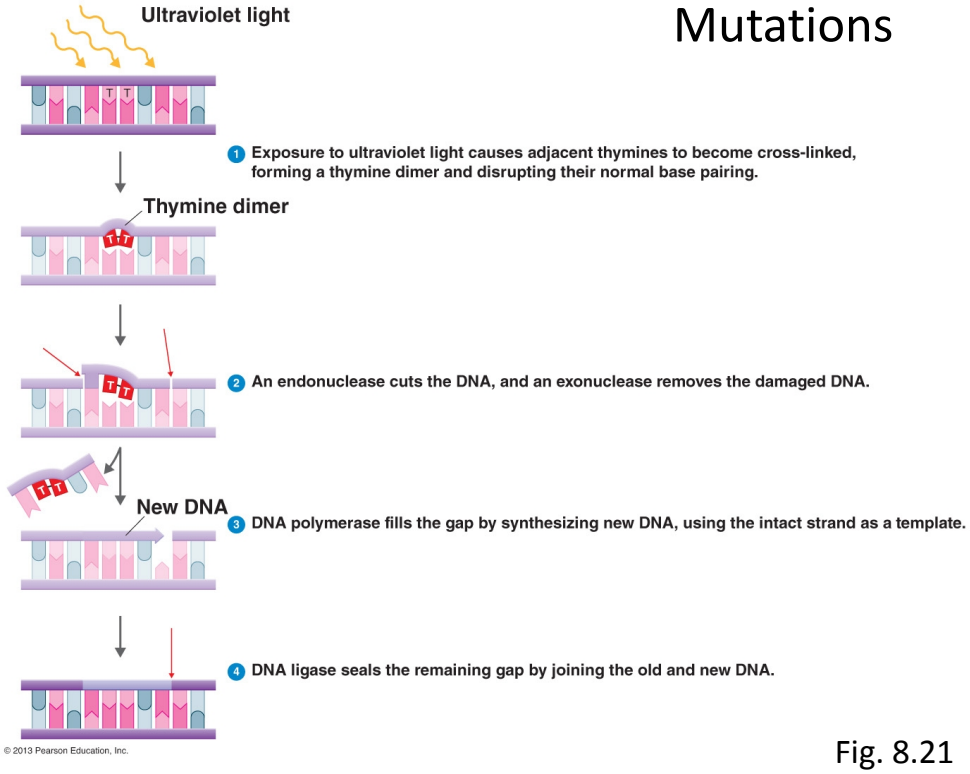
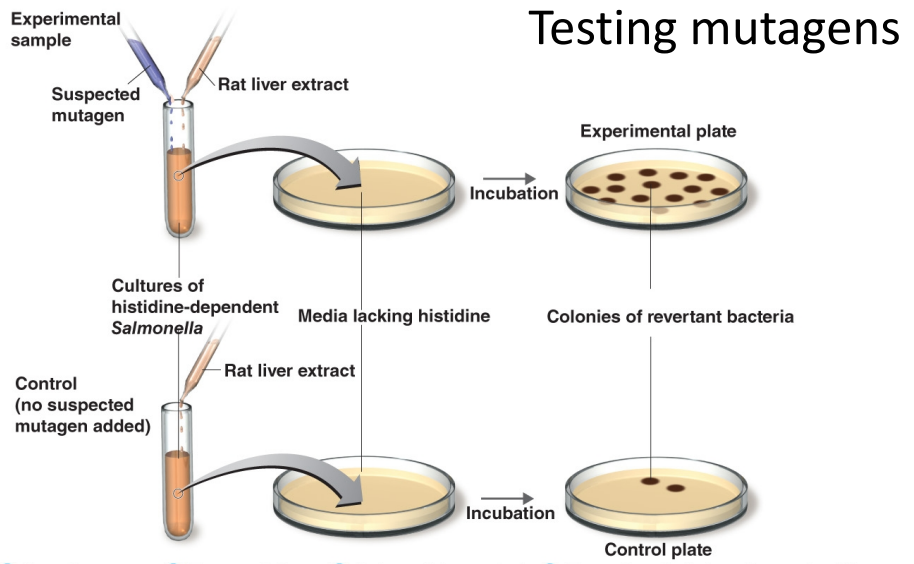


Fig. 8.21

Testing mutagens



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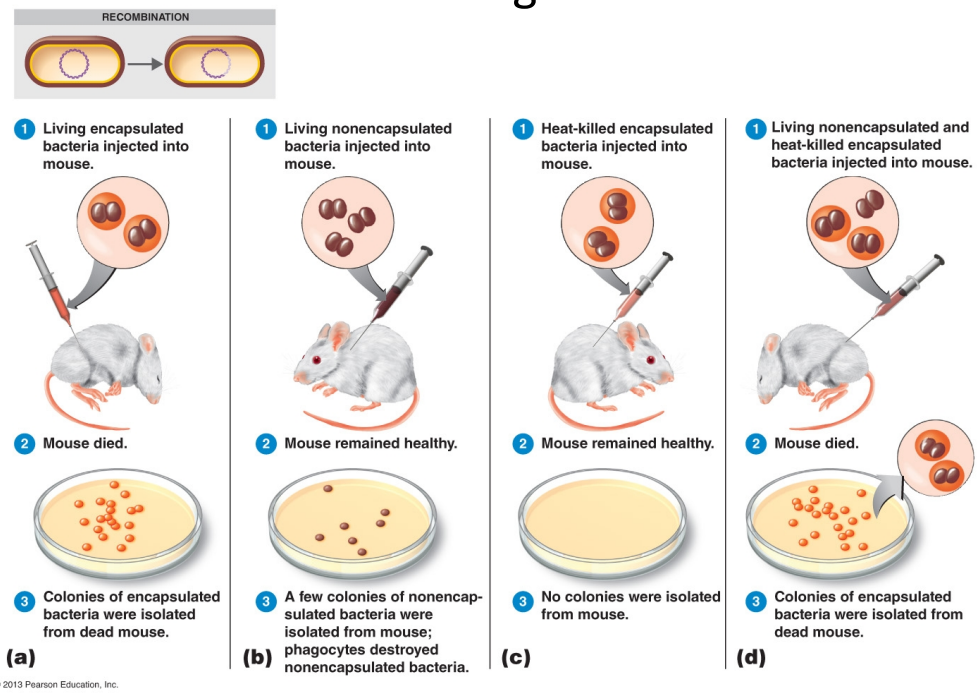


Fig. 8.25

Recombination: Transformation

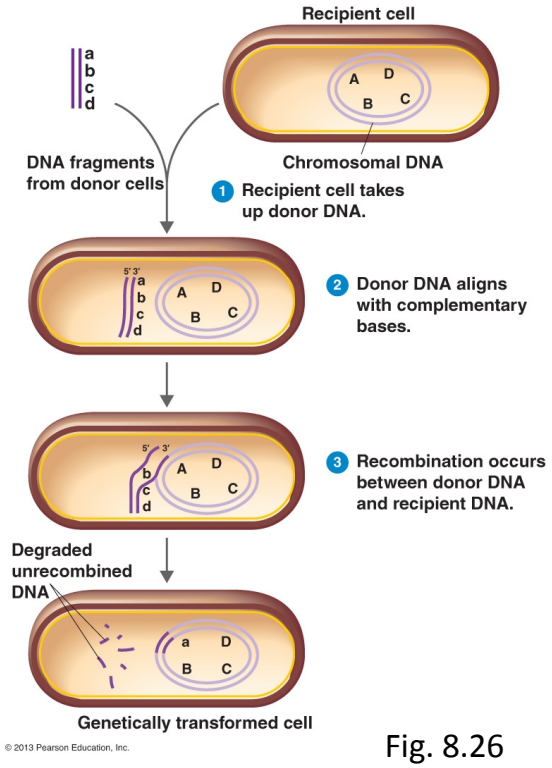
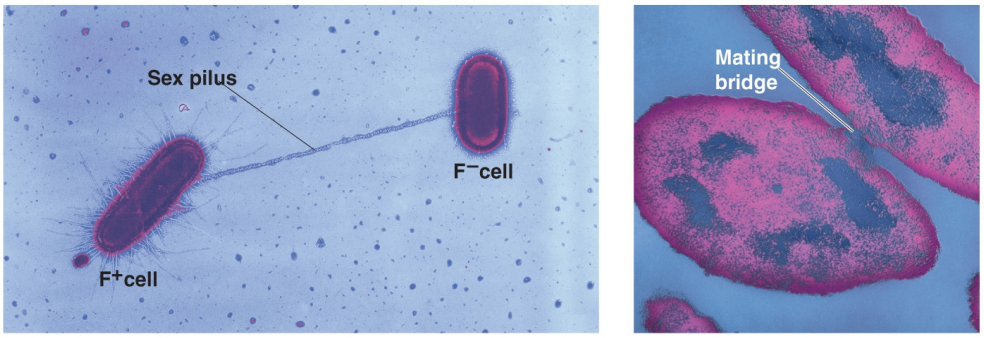


Fig. 8.26

Recombination: Conjugation



(a) Sex pilus TEM 1 μm

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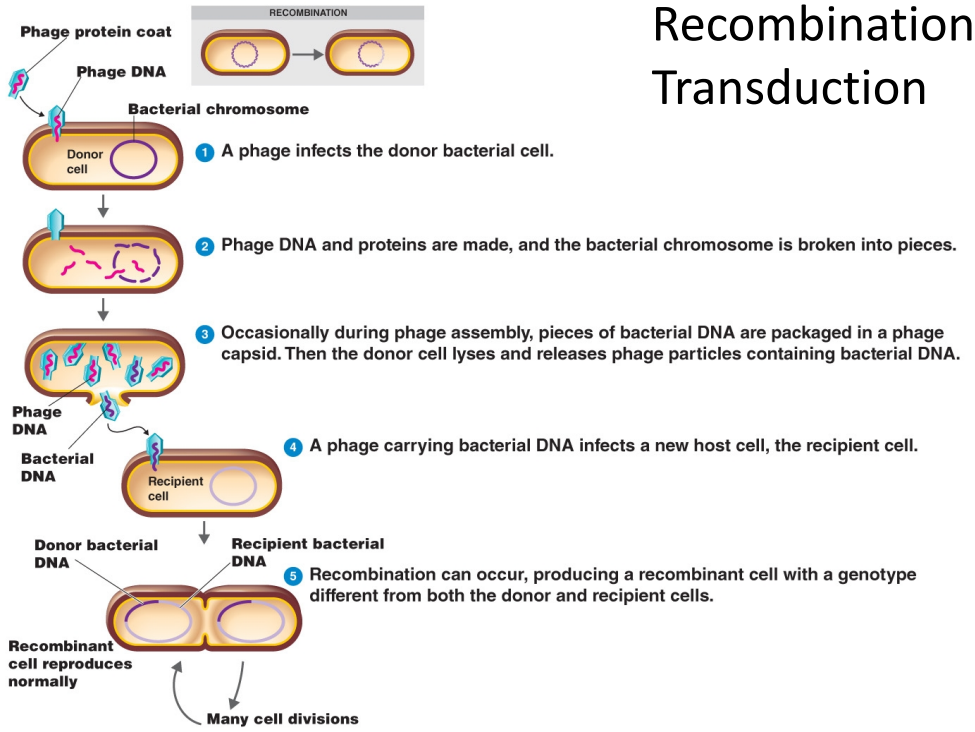

Fig. 8.29

Plasmids

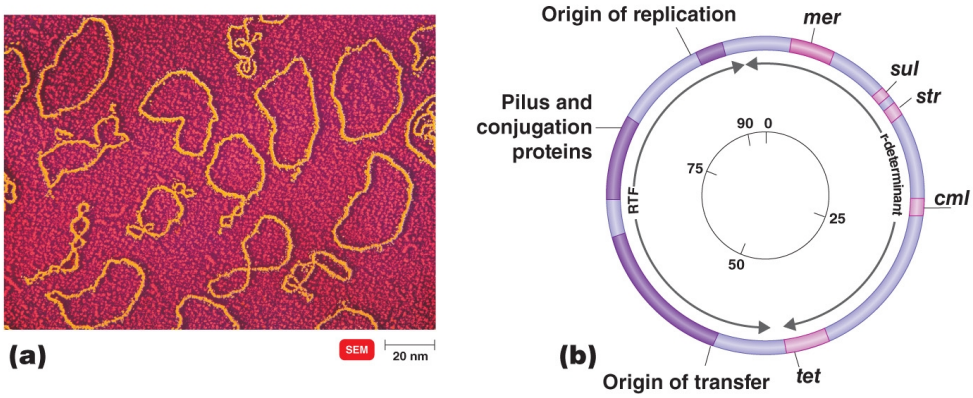

Fig. 8.30

Operon model

Fig. 8.13

Operon model

Fig. 8.13