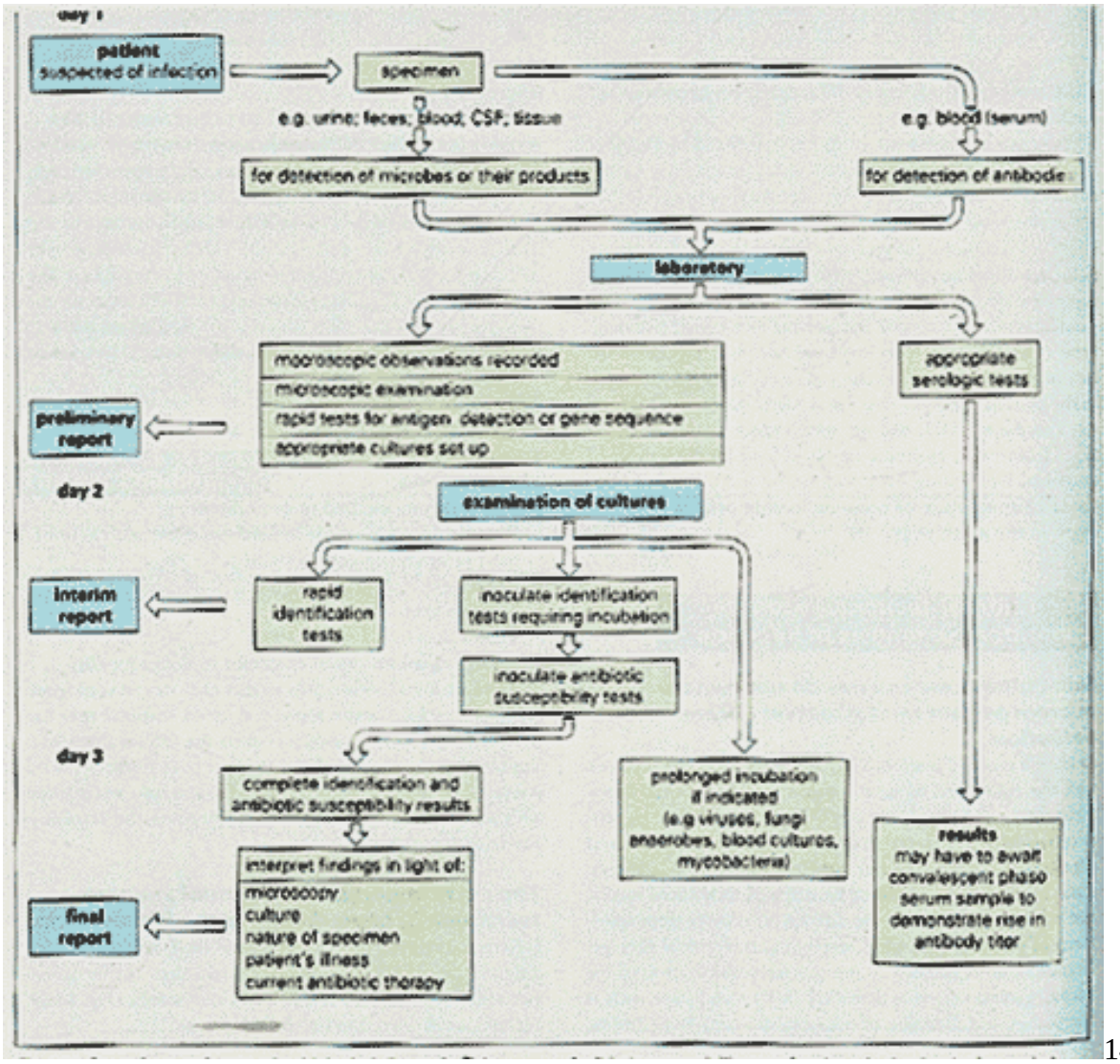

day 1
patient
suspected of infection
specimen
e.g. urine; feces; blood; CSF; tissue
e.g. blood (serum)
for detection of microbes or their products
for detection of antibodie
laboratory
macroscopic observations recorded
microscopic examination
rapid tests for antigen detection or gene sequence
appropriate cultures set up
appropriate serologic tests
preliminary report
day 2
examination of cultures
interim report
rapid identification tests
inoculate identification tests requiring incubation
inoculate antibiotic susceptibility tests
day 3
complete identification and antibiotic susceptibility results
prolonged incubation if indicated (e.g viruses, fungi anaerobes, blood cultures, mycobacteria)
results may have to await convalescent phase serum sample to demonstrate rise in antibody titer
interpret findings in light of:
microscopy
culture
nature of specimen
patient's illness
current antibiotic therapy
final report
14a

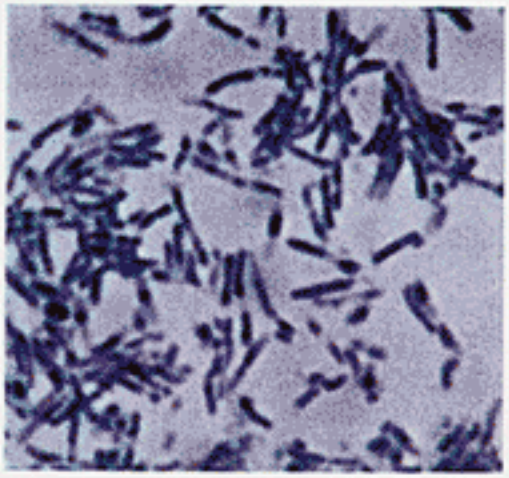


Figure 190
Metachromatic granules (enlarged and darkly stained areas) of *Corynebacterium diphtheriae.* (1,000×).

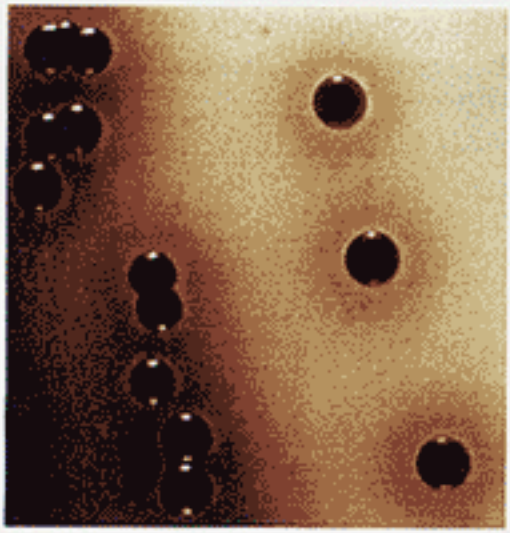


Figure 191
Ccolonies of *Corynebacterium diphtheriae* on a tellurite-containing medium.

14b

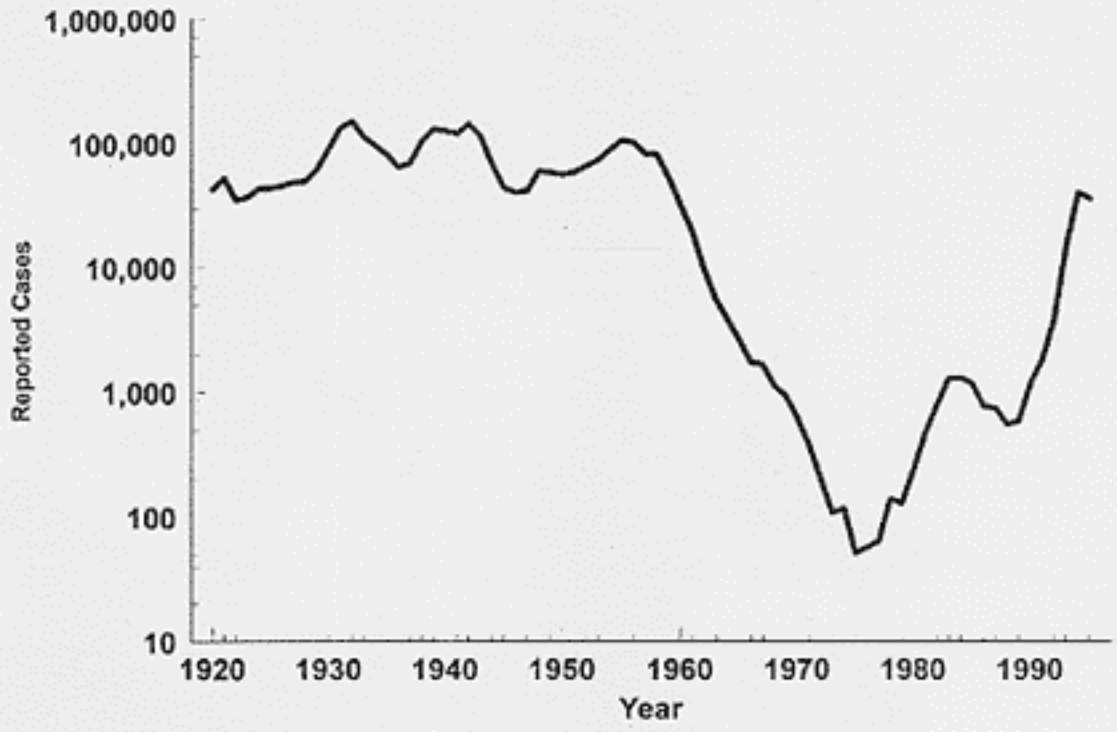
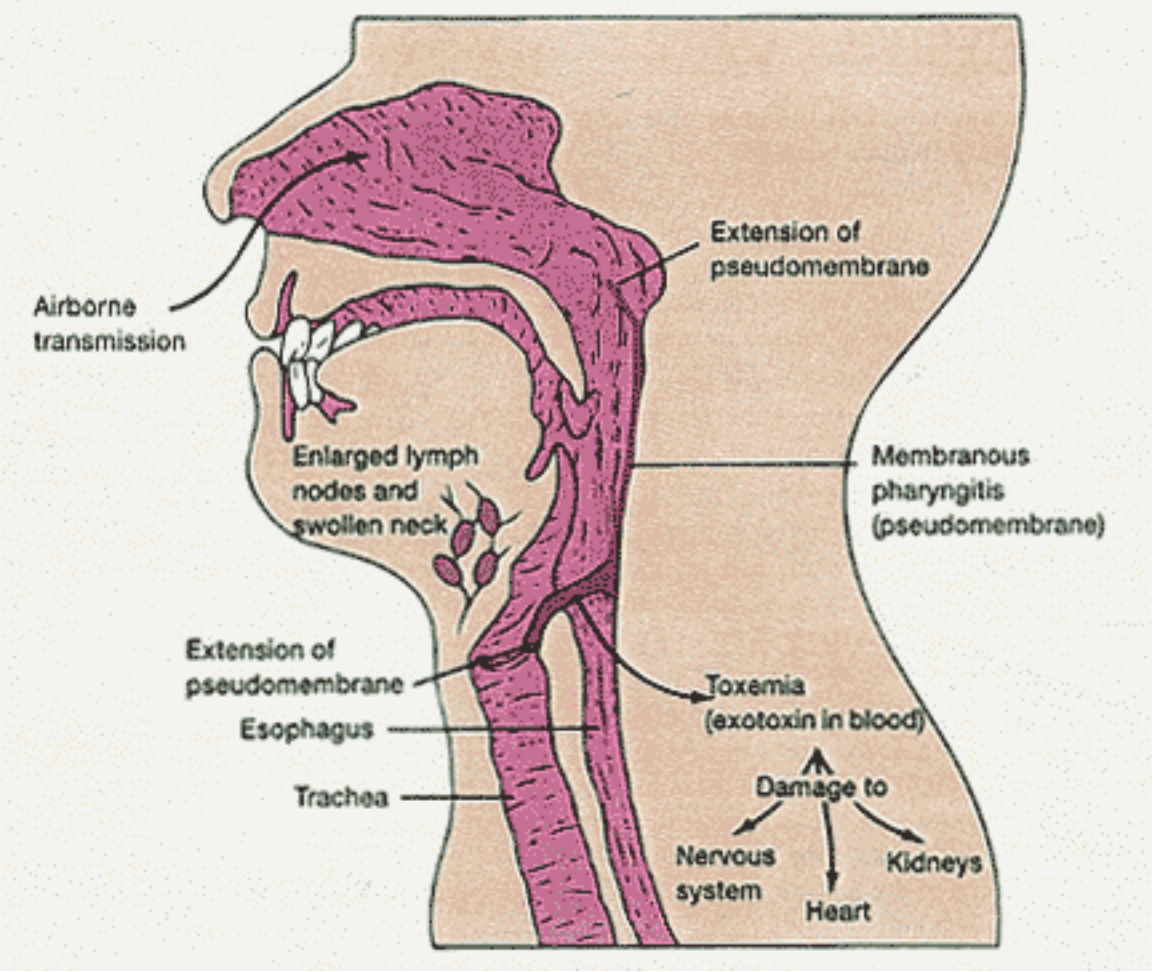
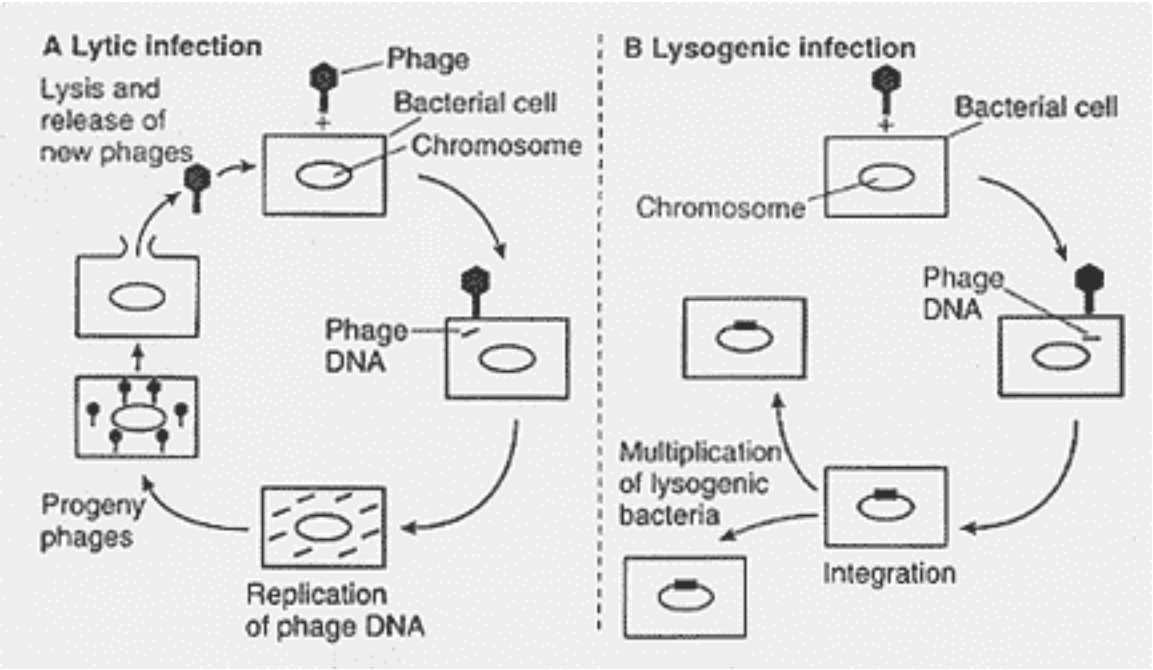


Figure 1. Reported cases of diphtheria in the Russian Federation from 1920 to 1995.

14c


Airborne transmission
Extension of pseudomembrane
Enlarged lymph nodes and swollen neck
Membranous pharyngitis (pseudomembrane)
Extension of pseudomembrane
Esophagus
Trachea
Toxemia (exotoxin in blood)
Damage to
Nervous system
Heart
Kidneys

14d


A Lytic infection
Lysis and release of new phages
Phage
Bacterial cell
Chromosome
Phage DNA
Progeny phages
Replication of phage DNA
B Lysogenic infection
Bacterial cell
Chromosome
Phage DNA
Multiplication of lysogenic bacteria
Integration

14e

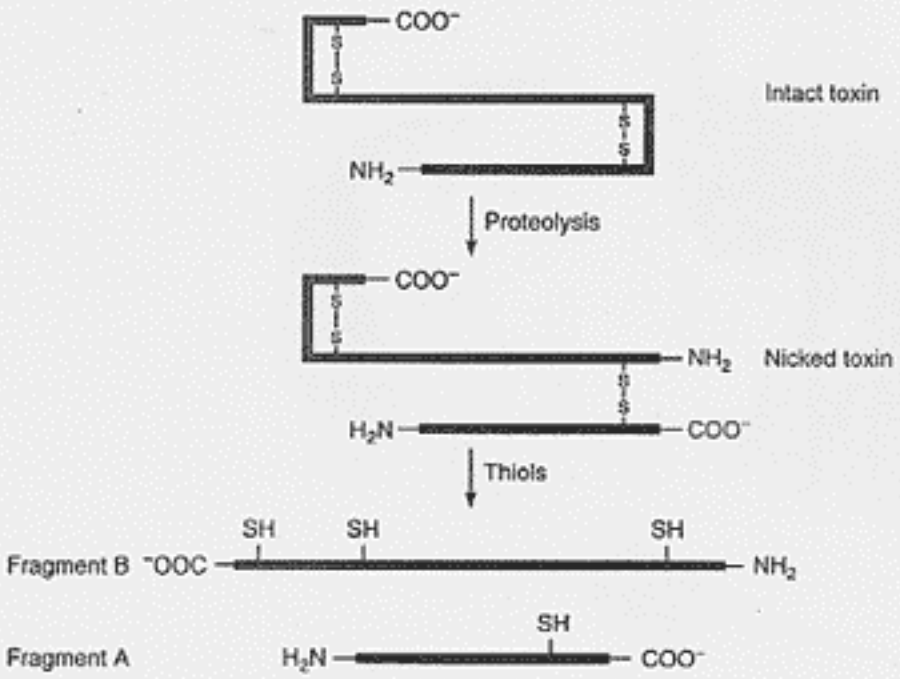
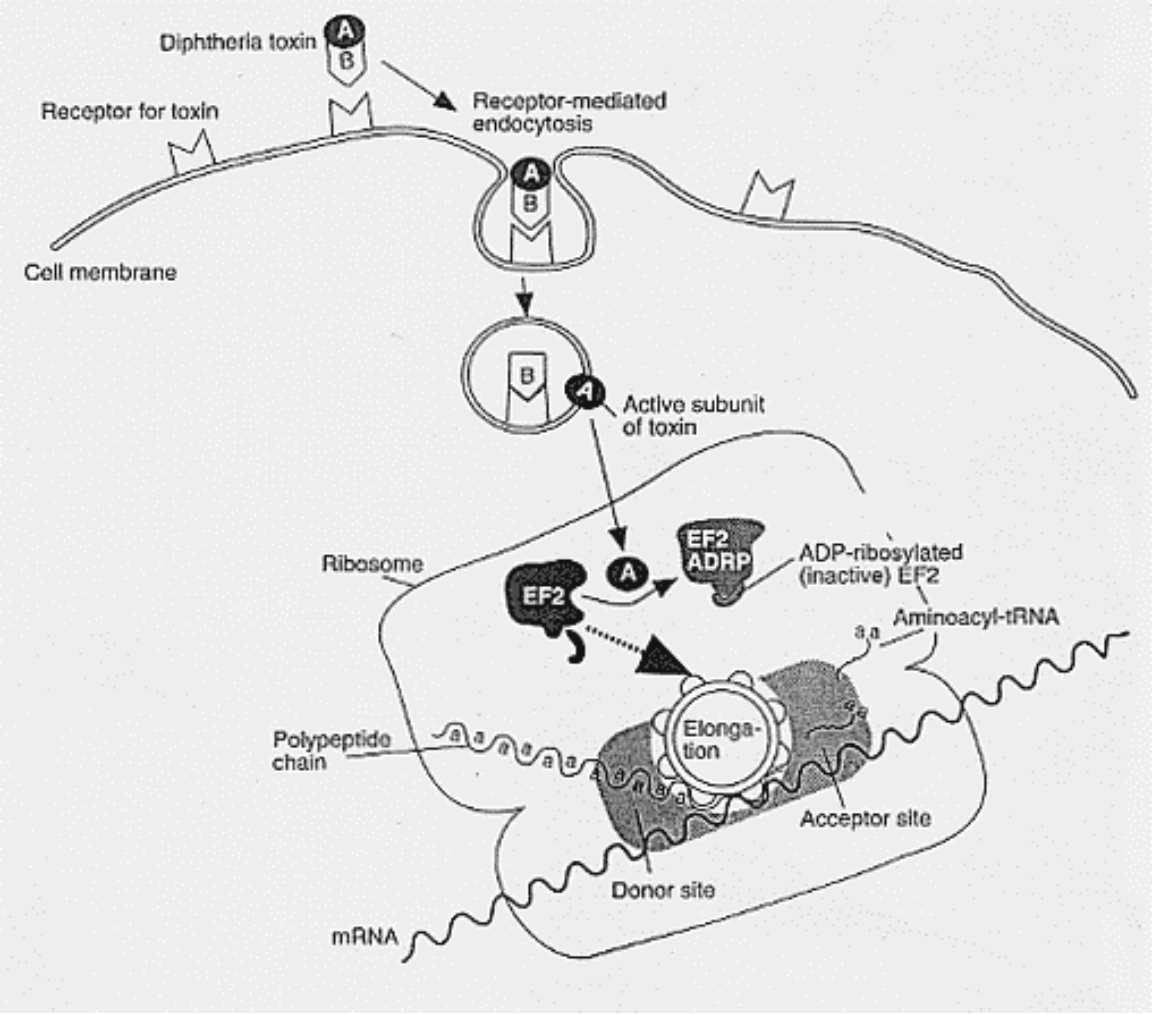


Fig. 8.1 Diphtheria toxin production and post-translation modification. The toxin is synthesised as a single polypeptide but is cleaved (nicked) by proteases into two fragments designated A and B, held together by an –S–S– bond. The latter is reduced probably during translocation of the A fragment into the cytosol.


Diphtheria toxin
A
B
Receptor for toxin
Receptor-mediated endocytosis
Cell membrane
Active subunit of toxin
Ribosome
EF2
EF2 ADRP
ADP-ribosylated (inactive) EF2
Aminoacyl-tRNA
Polypeptide chain
Elonga-tion
Acceptor site
Donor site
mRNA

14g

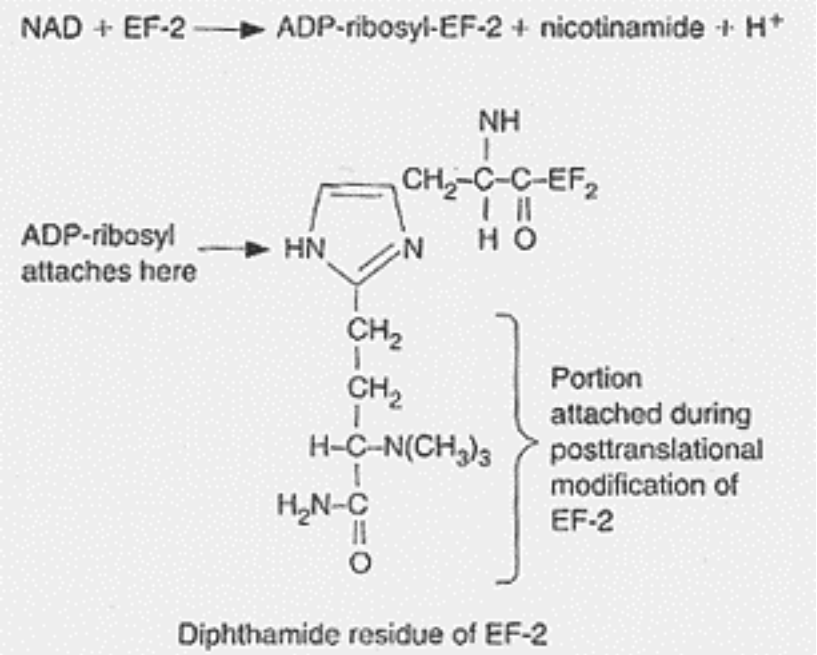
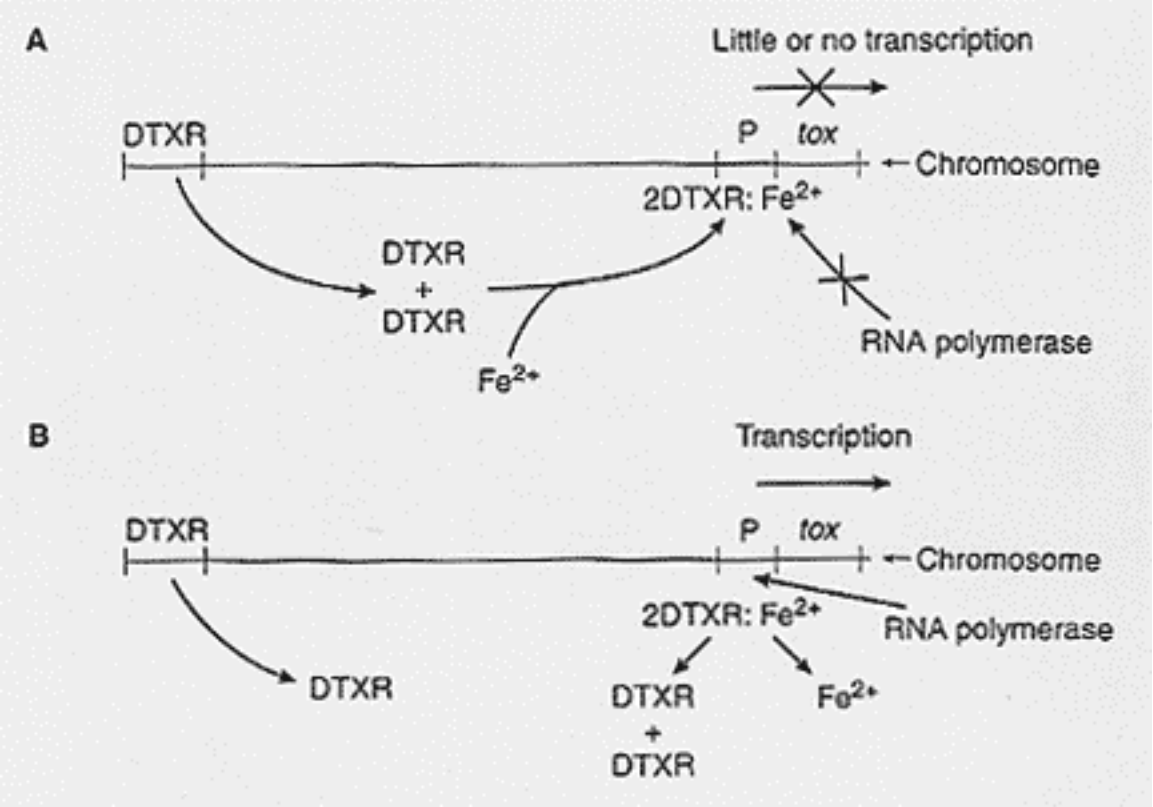


Figure 9-3 Reaction catalyzed by the A chain of diphtheria toxin and the structure of diphthamide, the modified histidine residue of EF-2 that is ADP-ribosylated.

14h



14i

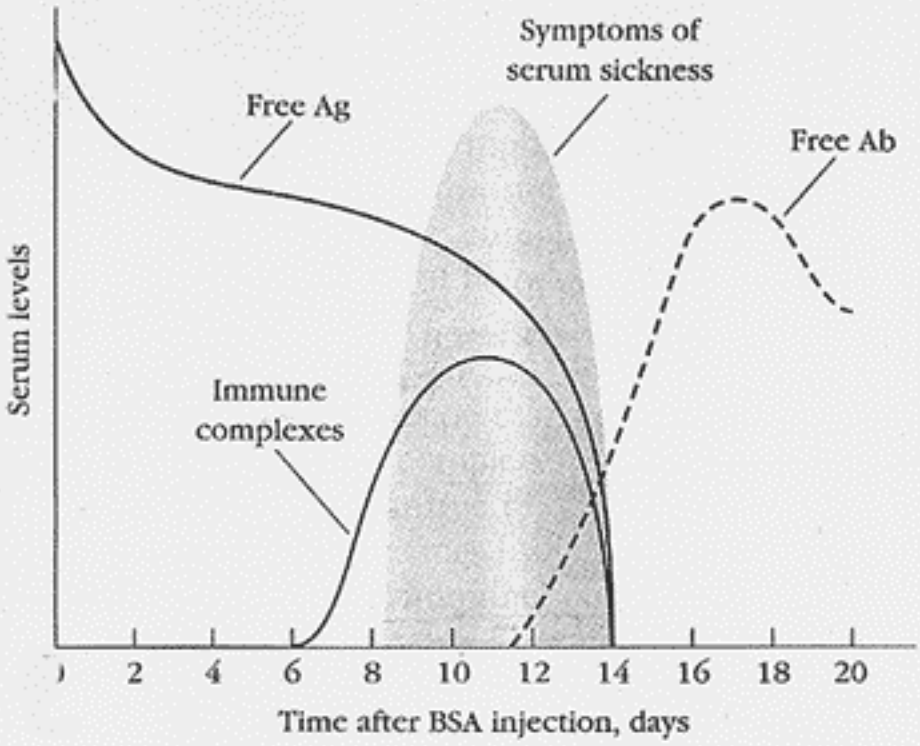


FIGURE 17-15

Correlation between immune-complex formation and development of symptoms of serum sickness. A large dose of antigen (BSA) was injected into a rabbit at day 0. As antibody formed, it complexed with the antigen and was deposited in the kidneys, joints, and capillaries. The symptoms of serum sickness (light blue curve) corresponded to the peak in immune-complex formation. As the immune complexes were cleared, free circulating antibody (dashed black curve) was detected and the symptoms of serum sickness subsided. [Based on F. G. Germuth, Jr., 1953, *J. Exp. Med.* 97:257.]

HYPERSENSITIVITY REACTIONS

Mediator	Type	Initiation Time	Reaction	Examples
Antibody	I (immediate, anaphylactic)	2-30 min	IgE antibody is induced by allergen and binds via its Fc receptor to mast cells and basophils. After encountering the antigen again, the fixed IgE becomes cross-linked, inducing degranulation and release of mediators (eg, histamine, serotonin, etc.)	Systemic anaphylaxis: Common allergens include: drugs (penicillin) and insect venom from bees and wasps) Localized anaphylaxis: Hayfever (upper respiratory) Asthma (lower respiratory) Food allergies (hives-skin eruptions)
Antibody	II (cytotoxic)	5-8 hours	Antigens on cell surface combine with antibody (IgM or IgG); this leads to complement-mediated lysis	Blood-transfusion reactions (ABO or Rh) Autoimmune hemolytic anemia
Antibody	III (immune complex)	2-8 hours	Large amounts of antigen-antibody immune complexes are deposited in tissues, complement is activated, and polymorpho-nuclear cells are attracted to the site, causing inflammation and tissue damage	Acute poststreptococcal glomerulonephritis Various autoimmune diseases (including rheumatoid arthritis, and systemic lupus erythematosus)
Cell	IV (delayed)	24 to 72 hours	T_H1 lymphocytes sensitized by antigen release cytokines that activate macrophages which, in turn, release various mediators which cause direct cellular damage.	Contact dermatitis to poison ivy and poison oak Tuberculin type hypersensitivity

14k