

Chapter 4

The infection and pathogenic mechanism of bacteria

Occurrence and development of bacterial infection

Origin of infection

1. Exogenous infection

(1) patient

(2) carrier

(3) animal

2. Endogenous infection

normal flora

The mode of infection

Horizontal transmission

person ↔ person ← animal
(zoonosis)

Vertical transmission

mother → offspring
placenta
birth canal
breast milk

The route of infection

- 1. respiratory tract**
- 2. digestive tract**
- 3. damaged skin or mucous membranes**
- 4. blood**
- 5. arthropod vector**
- 6. contact: sex contact (STD)**

The type and outcome of infection

I . inapparent infection

II . apparent infection

III . whole body infection

IV . carrier state

apparent infection

acute infection
chronic infection

local infection
systemic infection

**furuncle
carbuncle**

**bacteremia
toxemia
septicemia
pyemia**

whole body infection

toxemia: is the presence of exotoxin in the blood.

pyemia: is caused by purulent microorganisms in the blood.

endotoxemia: is the presence of endotoxin in the blood.

septicemia: illness that occurs when poisonous substances (toxins) produced by certain bacteria enter the bloodstream.

bacteremia: is an invasion of the bloodstream by bacteria.

Bacterial Pathogenesis Mechanism

☞ **virulence:**

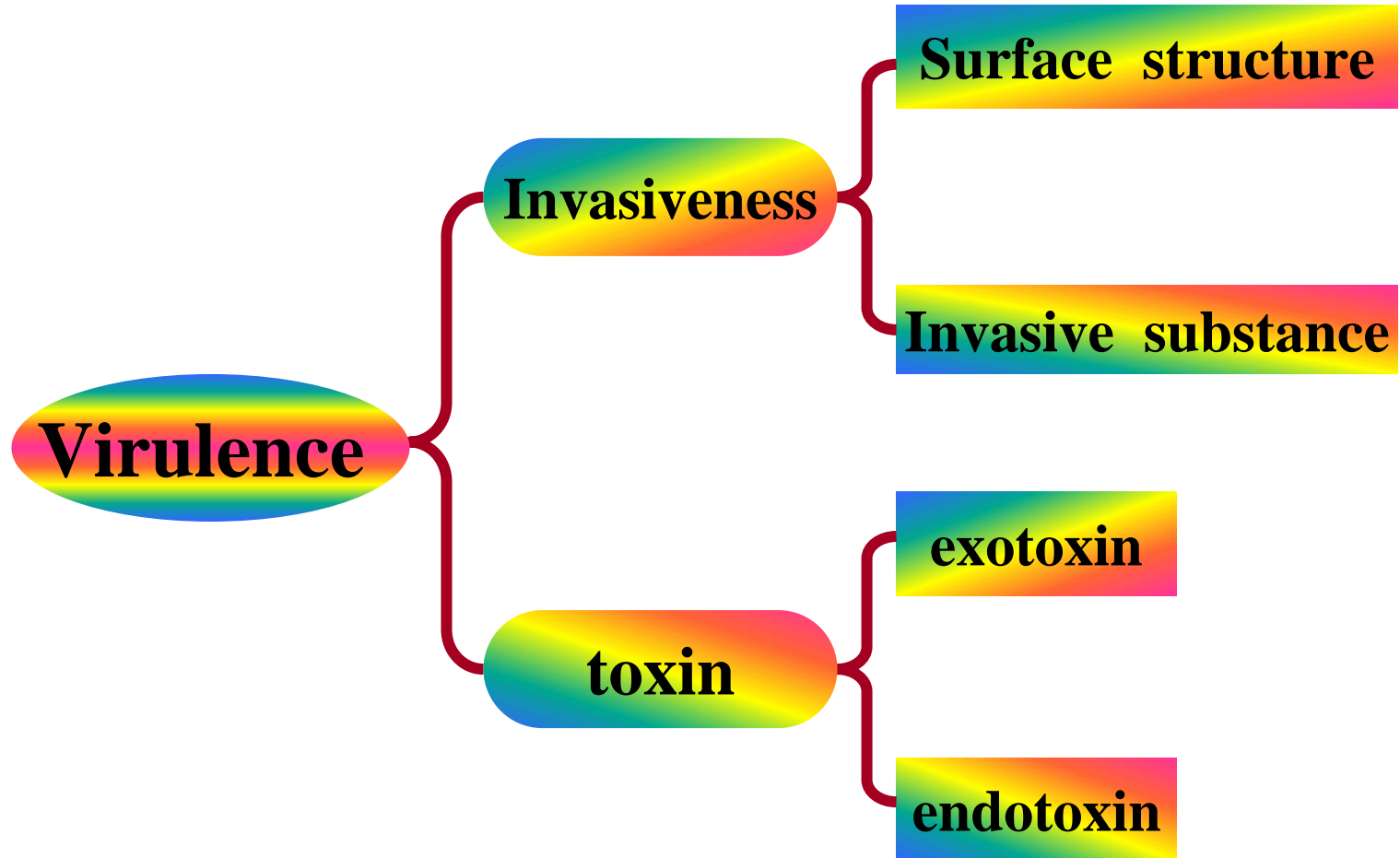
refers to extent of pathogenicity.

☞ **median lethal dose(LD50):**

The number of pathogens required to cause lethal disease in half of the exposed hosts is called an LD₅₀.

☞ **median infective dose(ID50):**

The number of pathogens required to cause disease (or, at least, infection) in half of the exposed hosts is called the ID₅₀.



(I) Invasiveness:

The ability of bacterium to resist host defence, colonize, multiply and spread.

1.adhesion -----surface structure

① bacteria to host cell:

pili

capsule

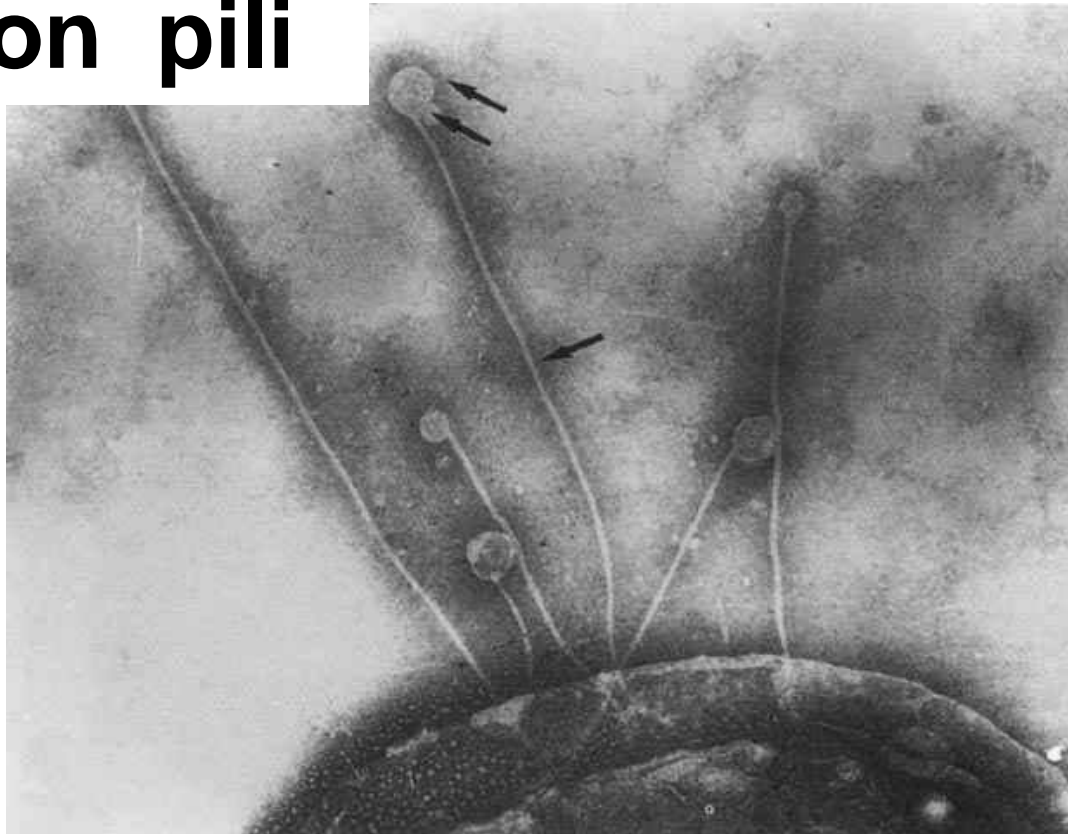
colonization factor

②bacteria to bacteria:

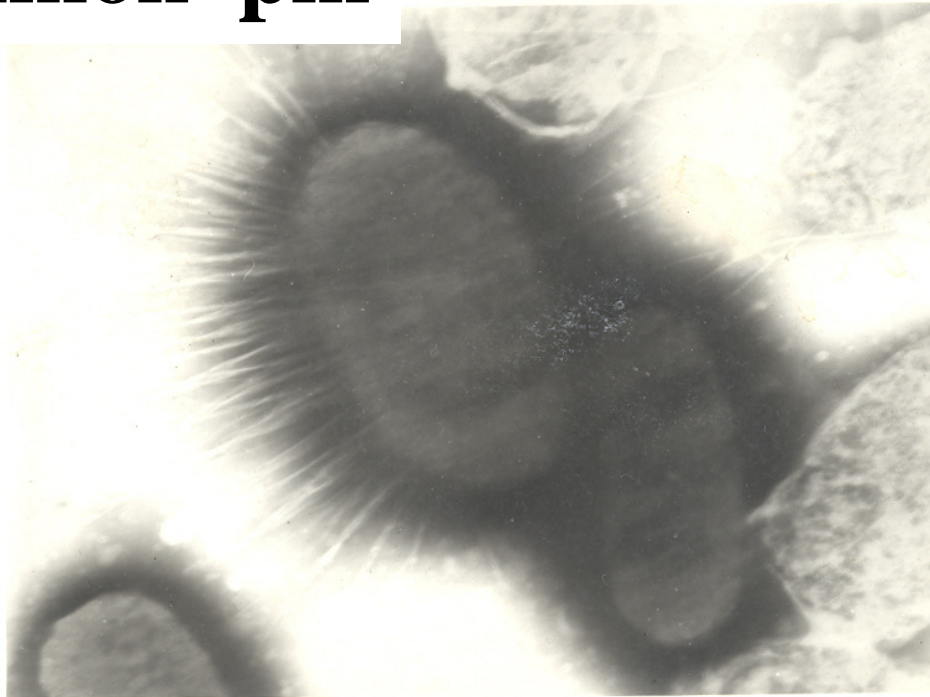
microcolony

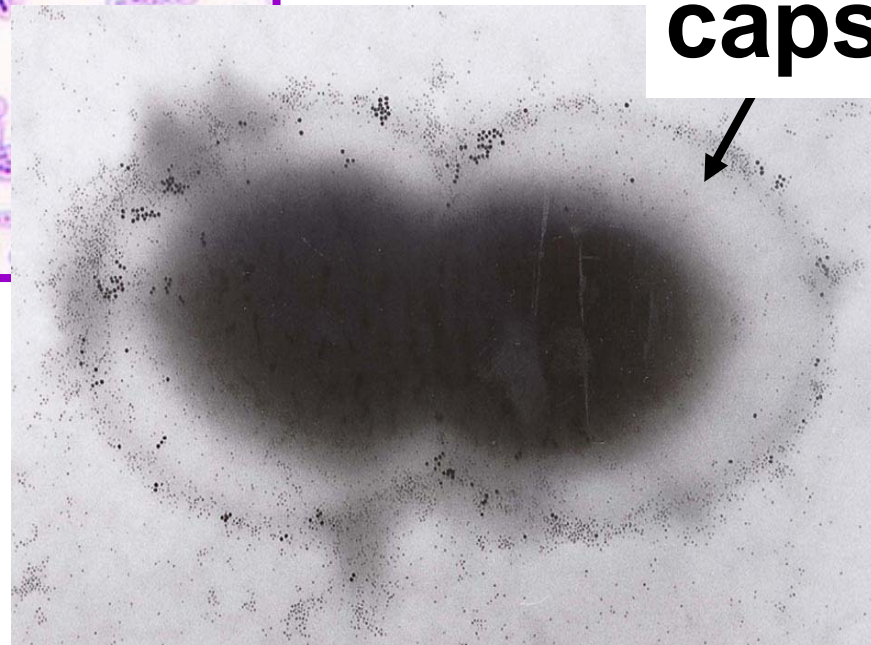
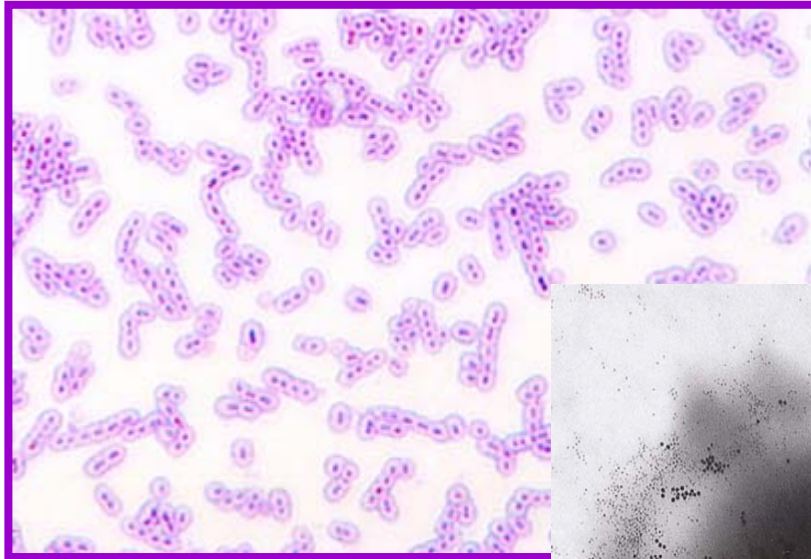
biofilm

common pili



common pili

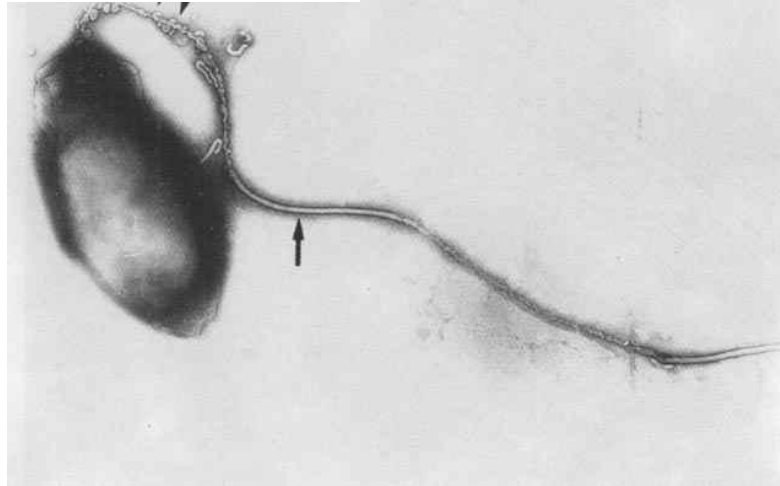




capsule



Flagella



2. Penetration and spread

capsule

invasive enzyme --- exoenzyme

- ***coagulase***--- *S. aureus*

fibrinogen → fibrin → surround bacteria

- ***hyaluronidase*** (spreading factor)

hydrolyze hyaluronic acid tissue loose, *B.* spreads

- ***streptokinase. SK*** ---- Lyse fibrin

- ***streptodornase, SD*** --- resolve DNA.

microcolony and biofilm

(II). toxin

1.exotoxin :

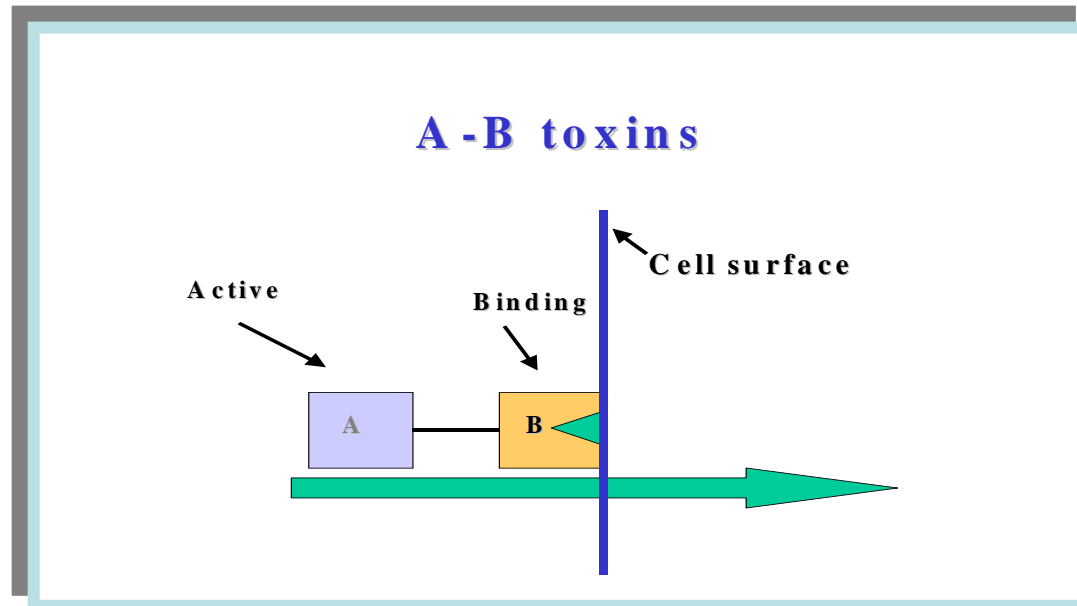
- **excreted by living cells, G⁺ bacteria**
- **polypeptide**
- **Heat-unstable, 60° C, 1-2hr destroy**
- **Strong antigenicity, exotoxin → toxoid**
- **highly toxic**
- **high selection for tissues**
- **structure : A subunit-B subunit**

neurotoxin
cytotoxin
enterotoxin

exotoxin

subunit A: toxicity active

subunit B: non-toxicity , bind receptor of sensitive cell



exotoxin

 **neurotoxin: tetanospasmin spinal cord**

 **cytotoxin: diphtherotoxin inhibit
cell protein synthesis**

 **enterotoxin: cholera toxin**





A severe case of tetanus.
muscles, back and legs are rigid
muscle spasms can break bones
can be fatal (e.g respiratory failure)



Clostridium tetani



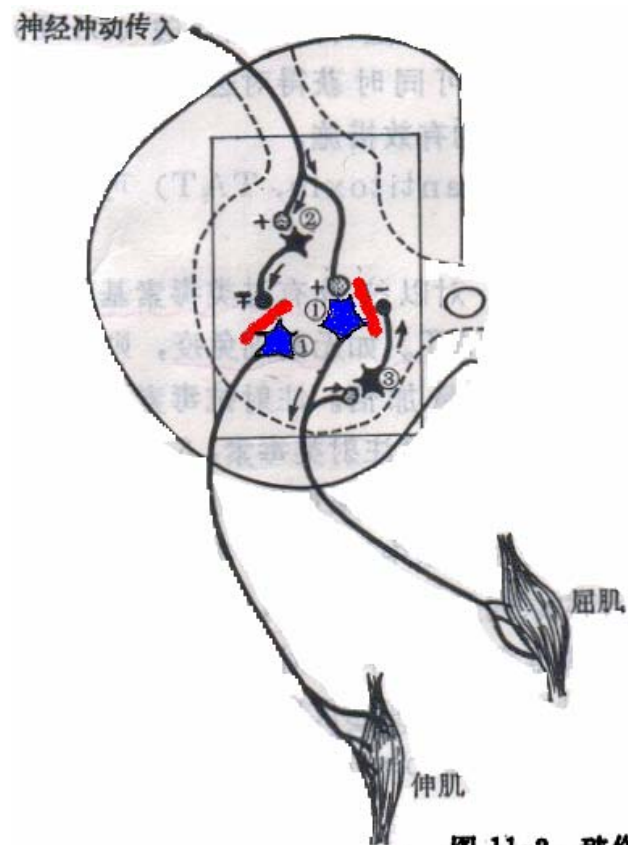
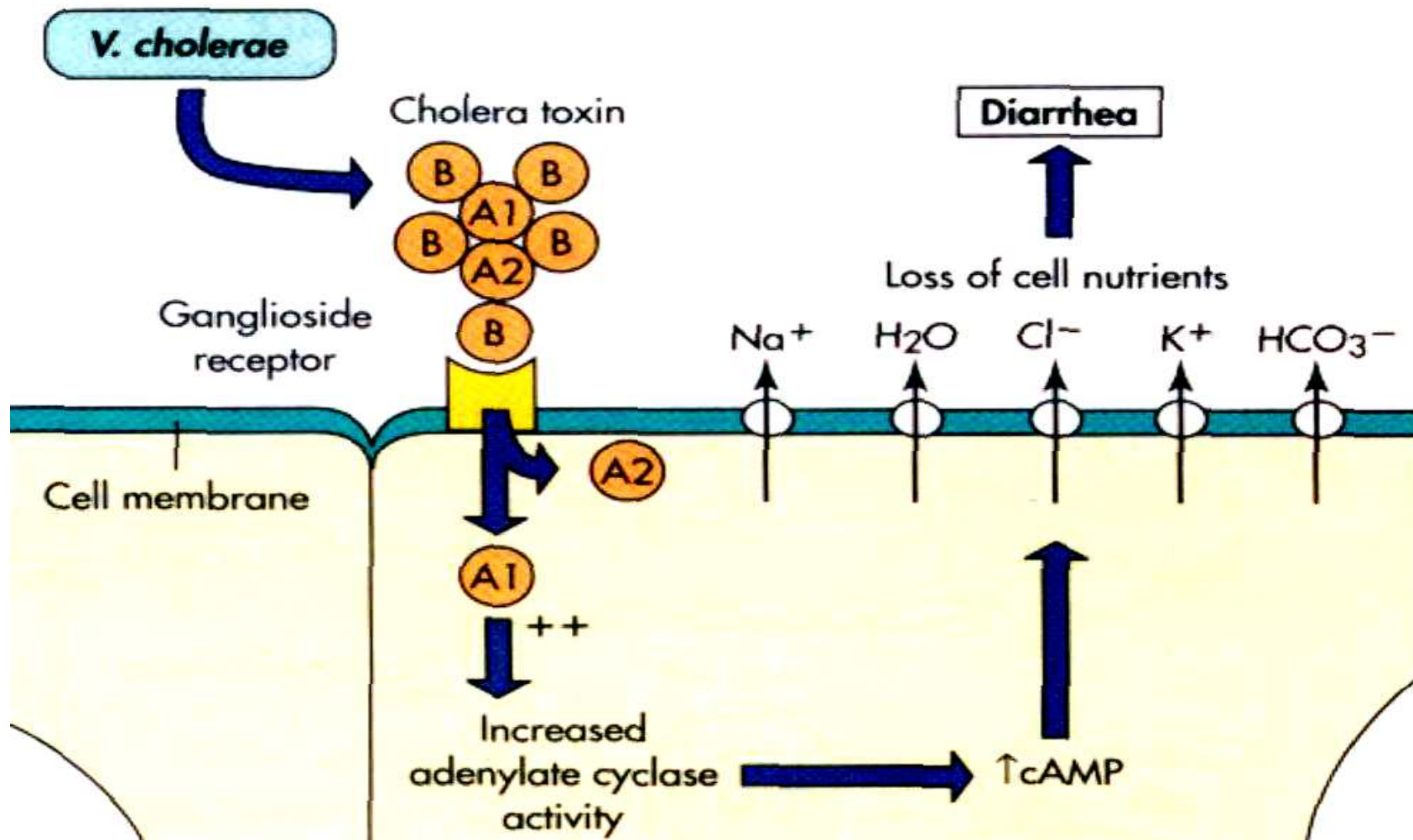


图 11-2 破伤风毒素的作用机理

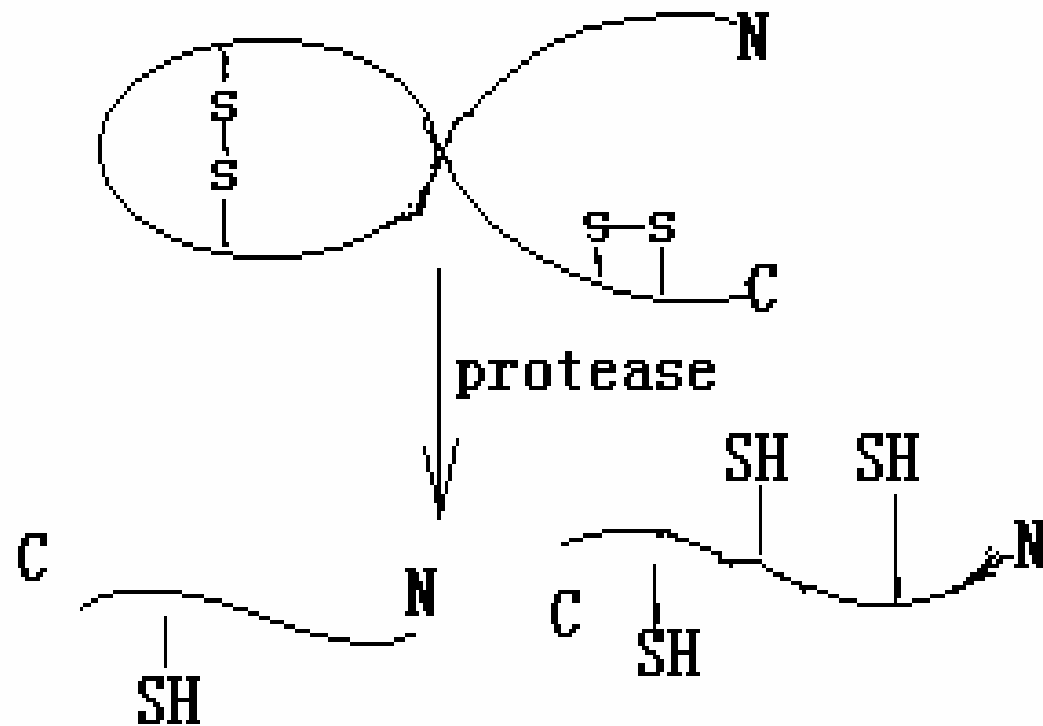
①运动神经元；②抑制性中间神经元；③Renshaw 细胞；+ 兴奋；- 抑制







This child has diphtheria resulting in a thick gray coating over back of throat. This coating can eventually expand down through airway and, if not treated, the child could die from suffocation



A fragment

B fragment

2. endotoxin

- **integral part of G⁻ bacteria cell wall.
release after bacteria disintegration**
- **LPS, main toxic part: Lipid A**
- **heat-stable: 160°C 2-4hr**
- **can't converted into toxoid**
- **weakly toxic**
- **non-specificity**

all endotoxins produce the same symptoms.

- ① fever ② WBC reaction ③ endotoxemia and shock
④ Schwartzman and DIC**

内毒素结构示意图

