



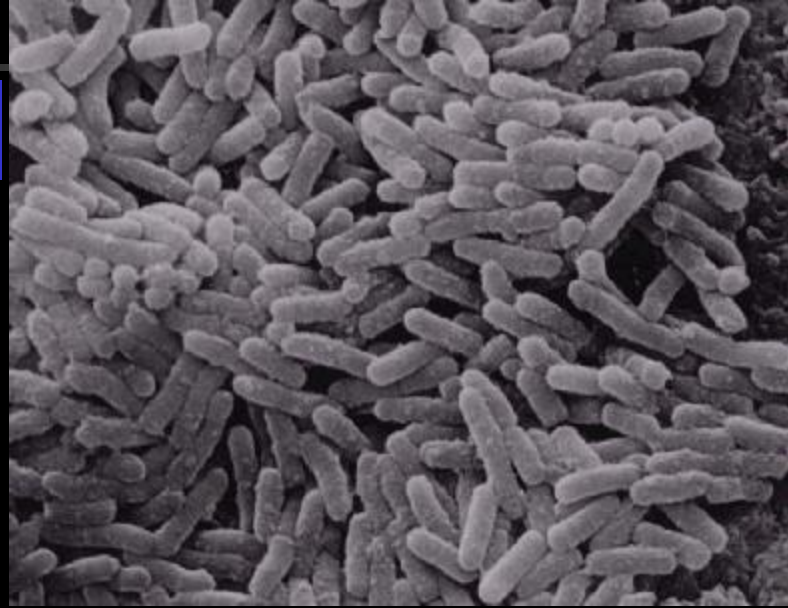
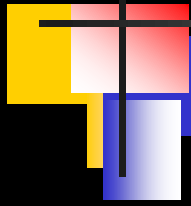
# **PRINCIPALES FACTORES DE VIRULENCIA BACTERIANA**

## **INVASIVIDAD**

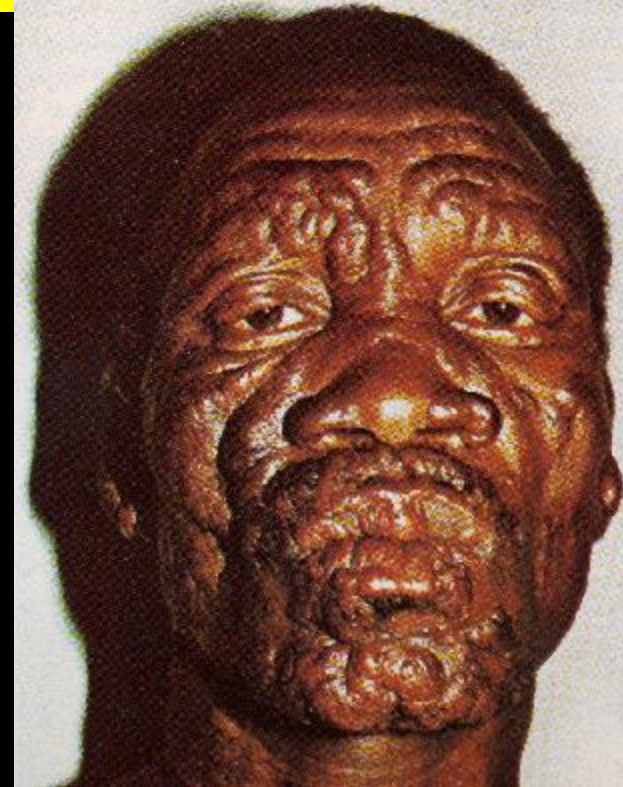
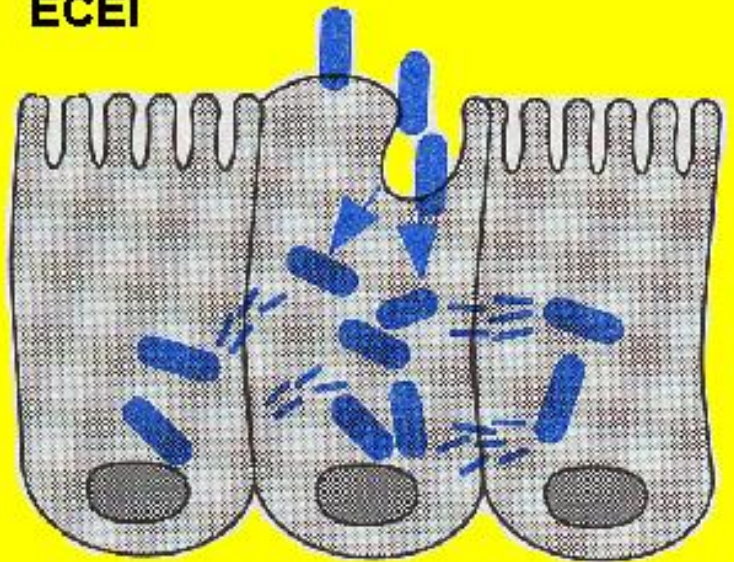
Capacidad de ciertas bacterias para establecerse, reproducirse y diseminarse en los tejidos del hospedero

## **TOXIGENICIDAD**

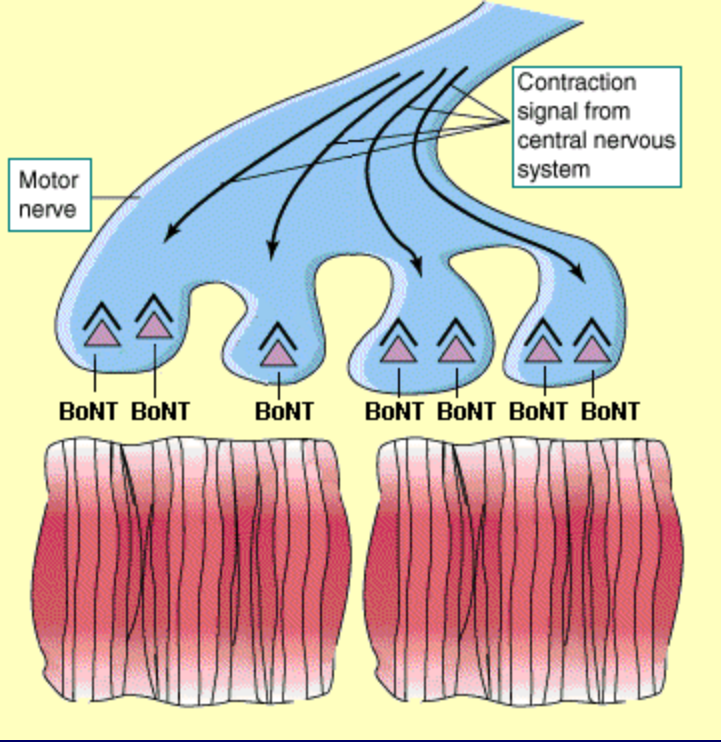
Capacidad de ciertas bacterias para producir toxinas



ECEI









# **MOLÉCULAS QUE SUSTENTAN LA INVASIVIDAD BACTERIANA**

**SUSTANCIAS ESTRUCTURALES,  
ENZIMAS Y MECANISMOS**

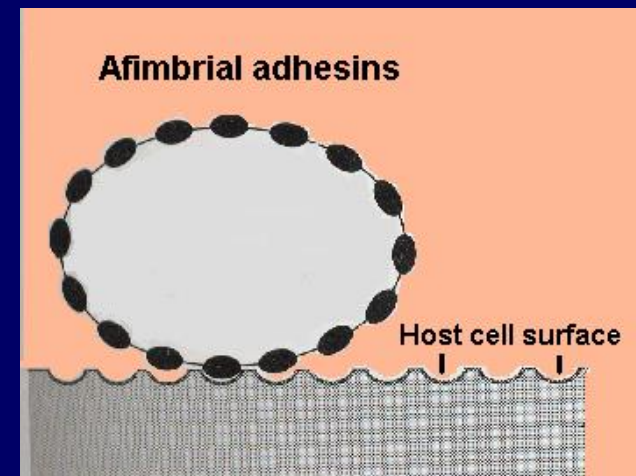
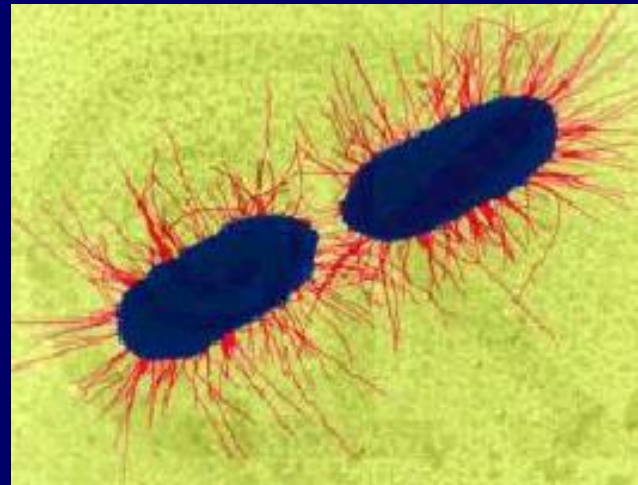
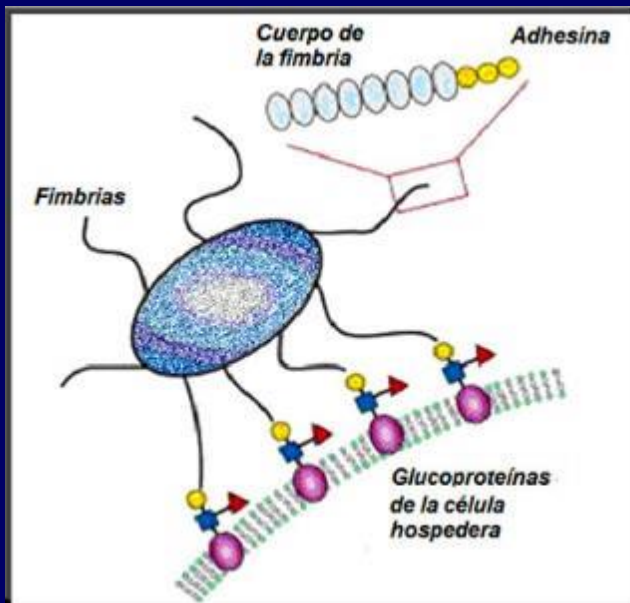


- **Establecimiento**
- **Reproducción-supervivencia**
- **Diseminación**

# MOLÉCULAS QUE SUSTENTAN LA INVASIVIDAD BACTERIANA

## Establecimiento: Adhesinas

### *Pili*/fimbrias



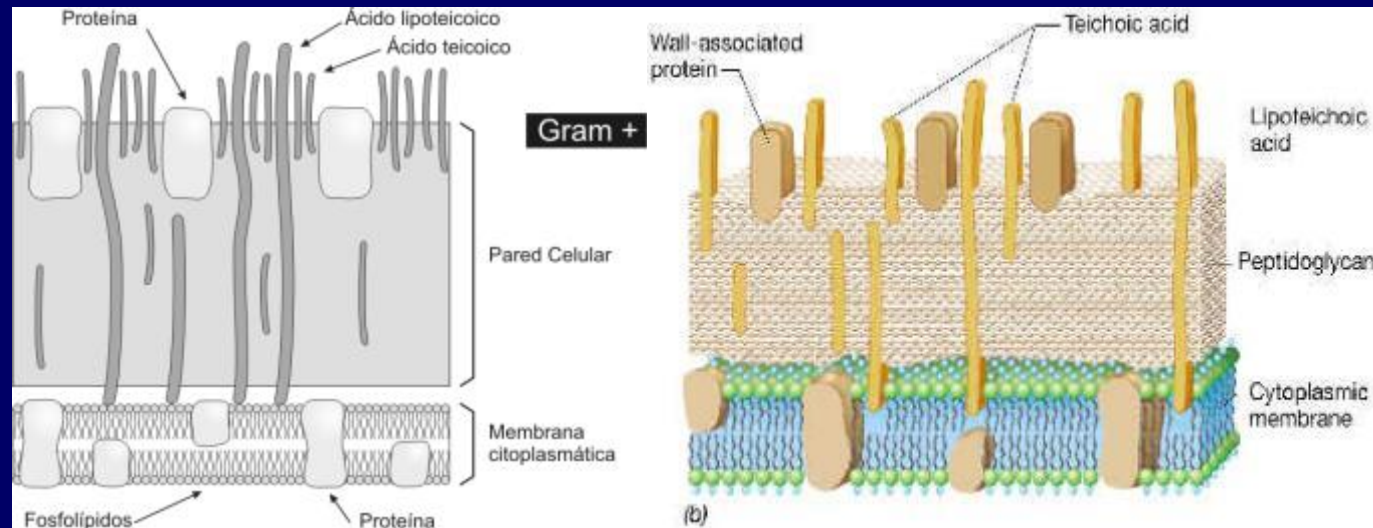
Proteínas no fimbriales



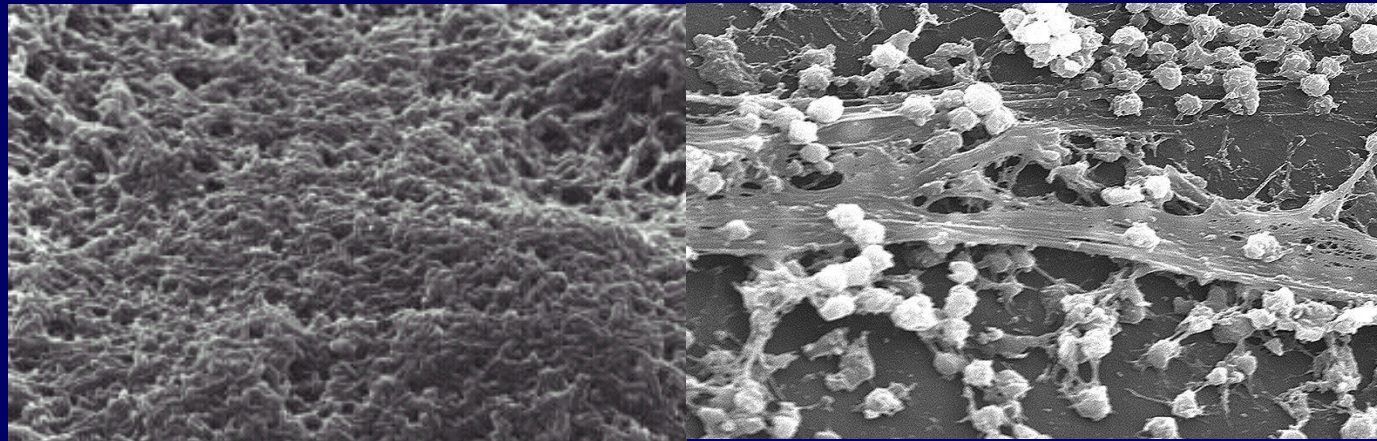
# MOLÉCULAS QUE SUSTENTAN LA INVASIVIDAD BACTERIANA

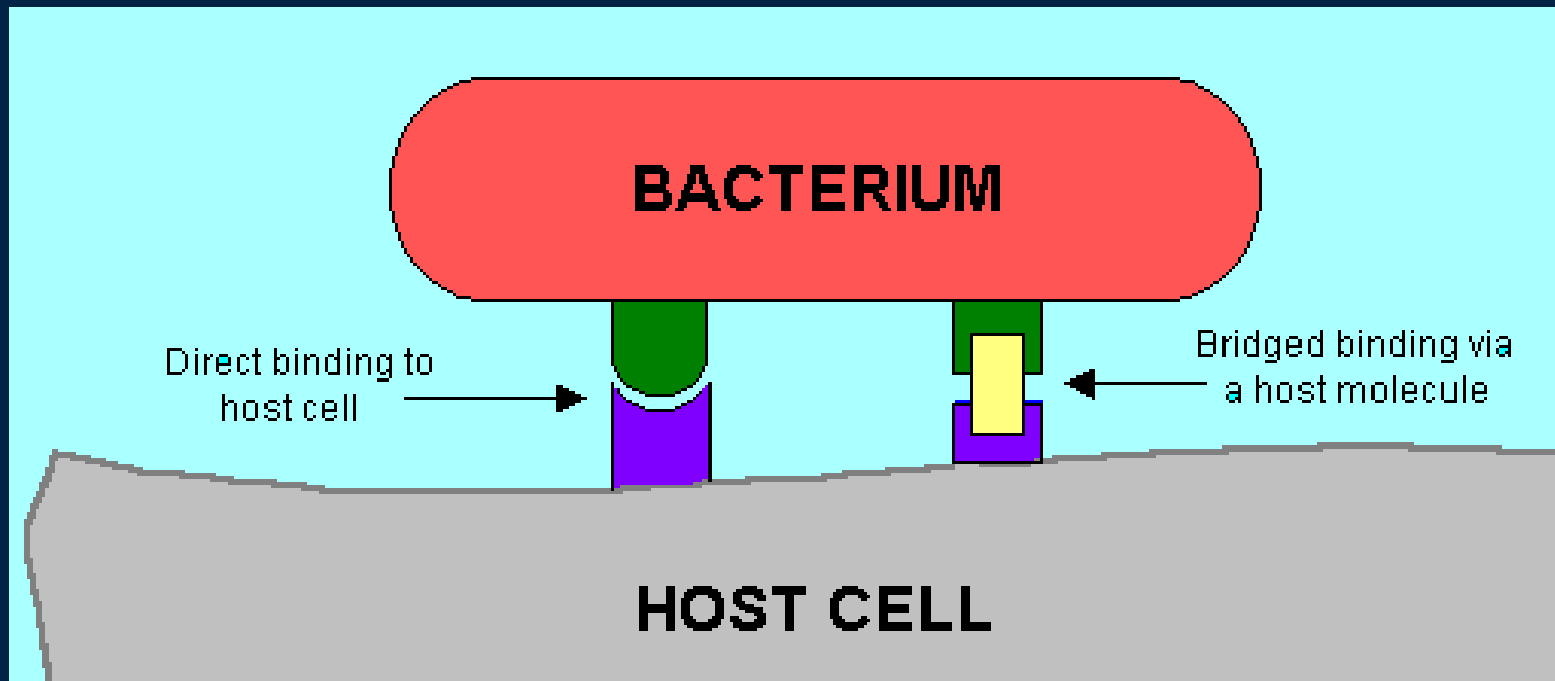
## Establecimiento: Adhesinas

Ácidos lipoteicoicos



Glicocálices



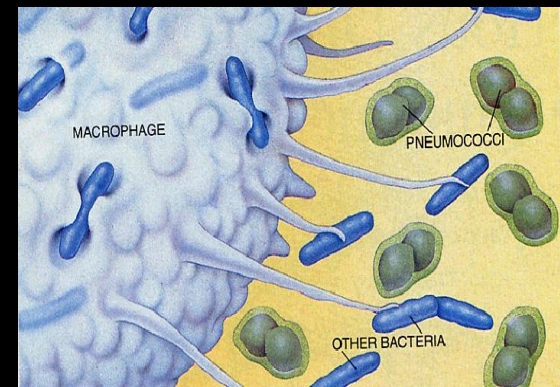


# MOLÉCULAS QUE SUSTENTAN LA INVASIVIDAD BACTERIANA

## Reproducción-supervivencia

### 1. Sustancias estructurales que interfieren la fagocitosis

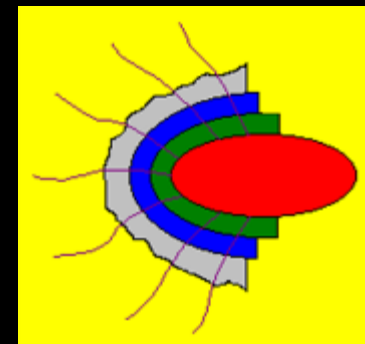
Cápsula



Periplasto

AgVi

Capa limosa

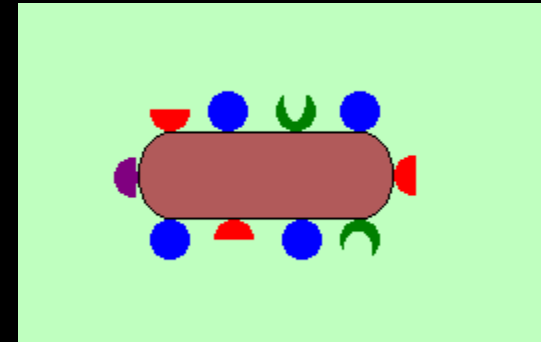




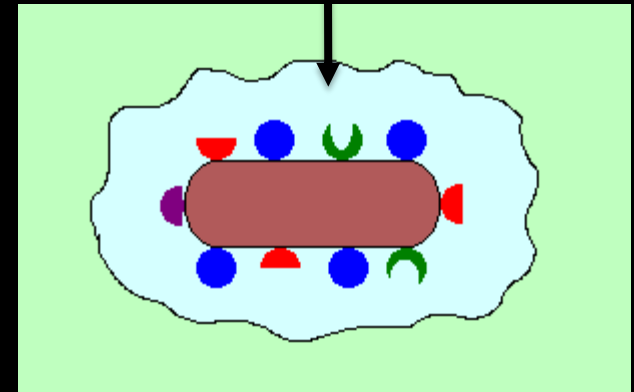
# Sustancias anti-fagocitosis

## Cápsulas, periplastos, AgVi, capas limosas

- Interfieren la fagocitosis
- Impiden la libre difusión de C3b, B, D, properdina, etc.
- Algunas son idénticas químicamente a sustancias del hospedero



Ácido hialurónico





# **MOLÉCULAS QUE SUSTENTAN LA INVASIVIDAD BACTERIANA**

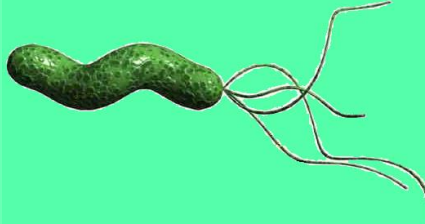
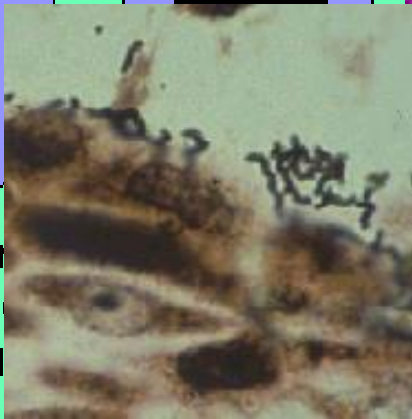
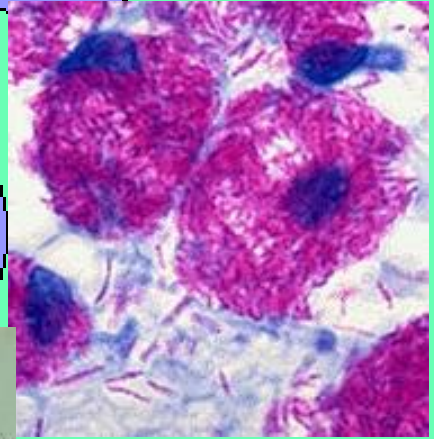
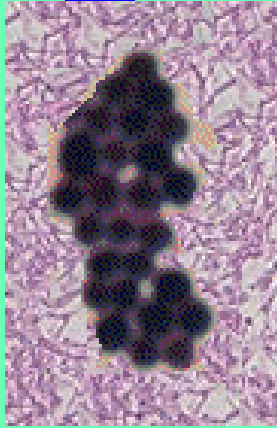
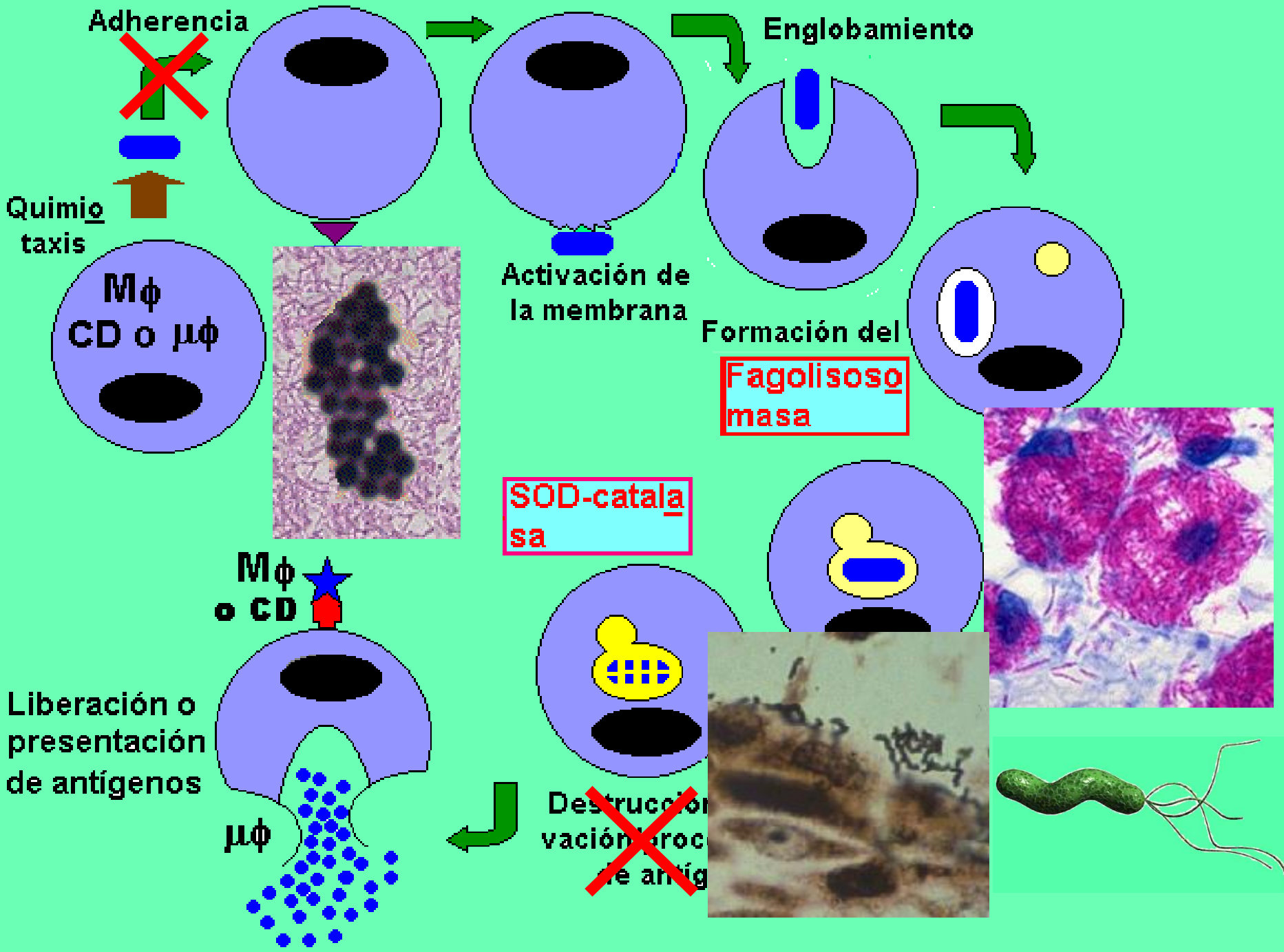
**Reproducción-supervivencia**

## **2. Enzimas que interfieren la fagocitosis**

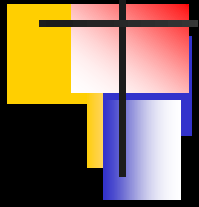
**Coagulasa**

**Fagolisosomasas**

**SOD-catalasa**

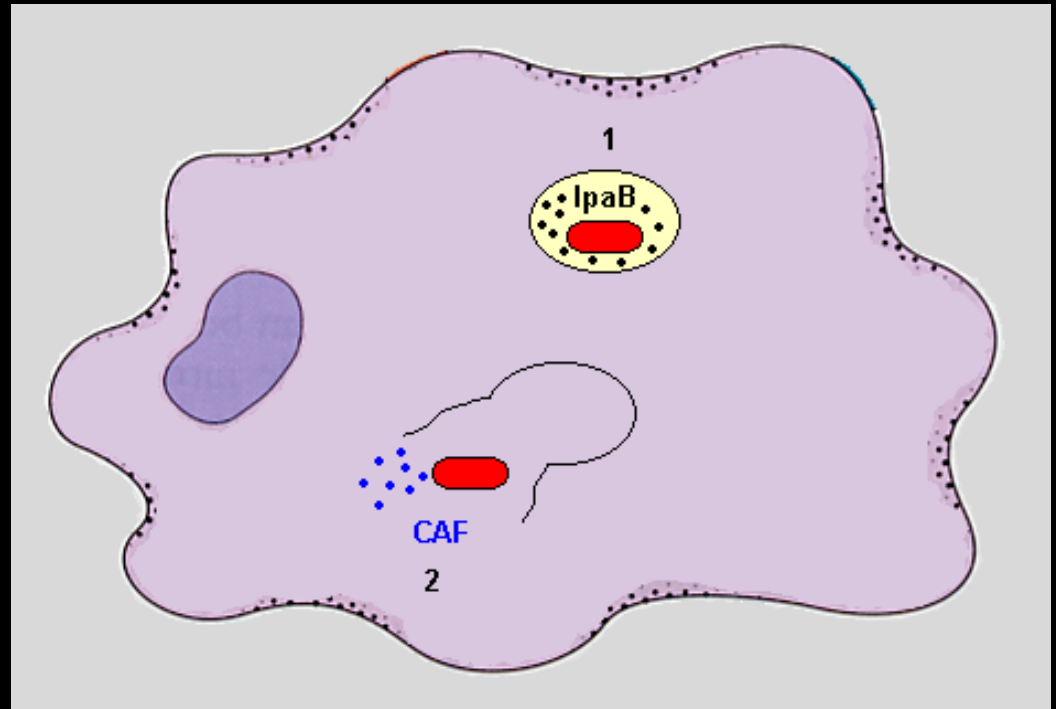






# Otras enzimas que interfieren la fagocitosis, originan:

- ✓ Escape de los fagosomas
- ✓ Inducción de apoptosis en el fagocito





# **MOLÉCULAS QUE SUSTENTAN LA INVASIVIDAD BACTERIANA**

## **Reproducción-supervivencia**

### **3. Enzimas que hidrolizan Acs o quimioatrayentes**

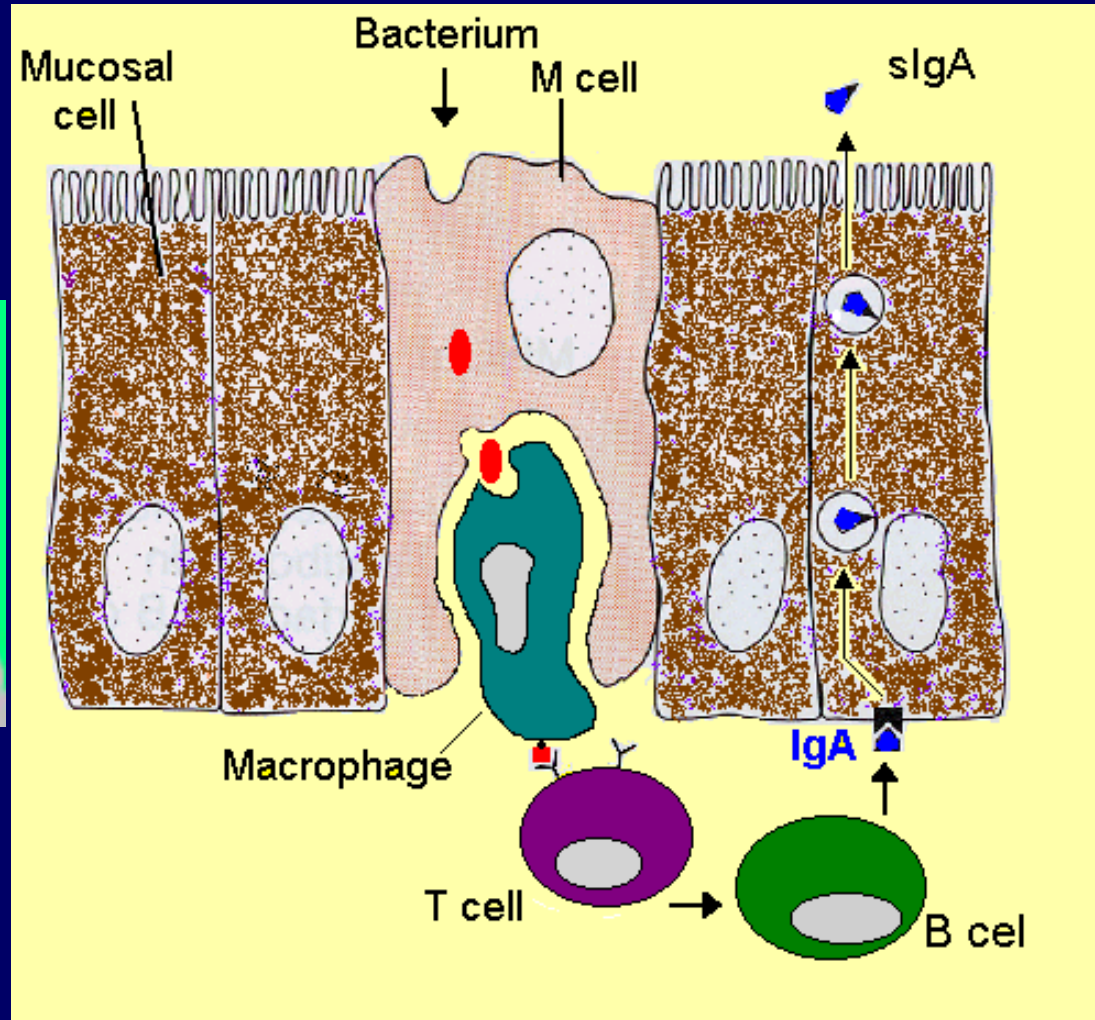
- ✓ **IgA hidrolasa**
- ✓ **C5a peptidasa**

### 3. Enzimas que hidrolizan Acs y quimioatrayentes

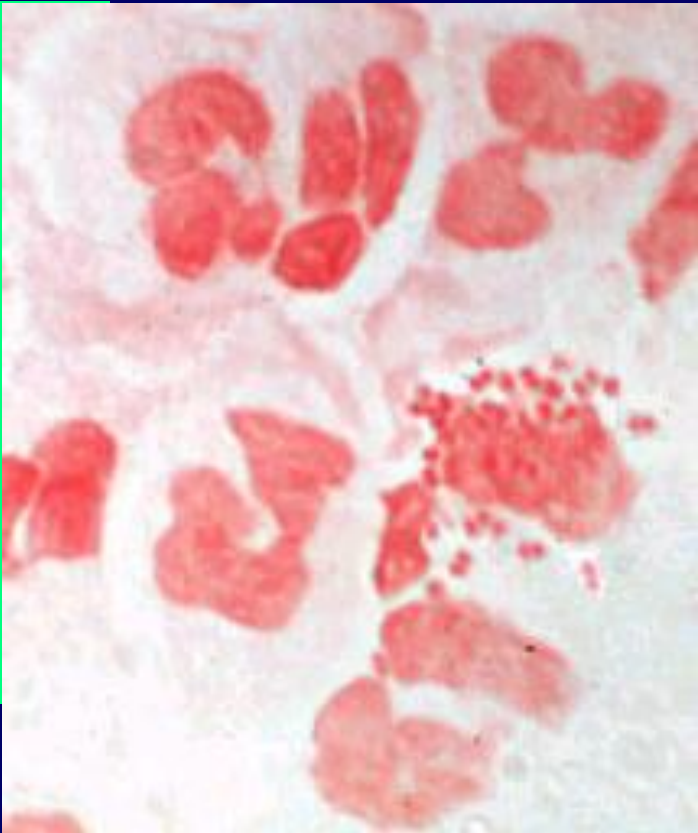
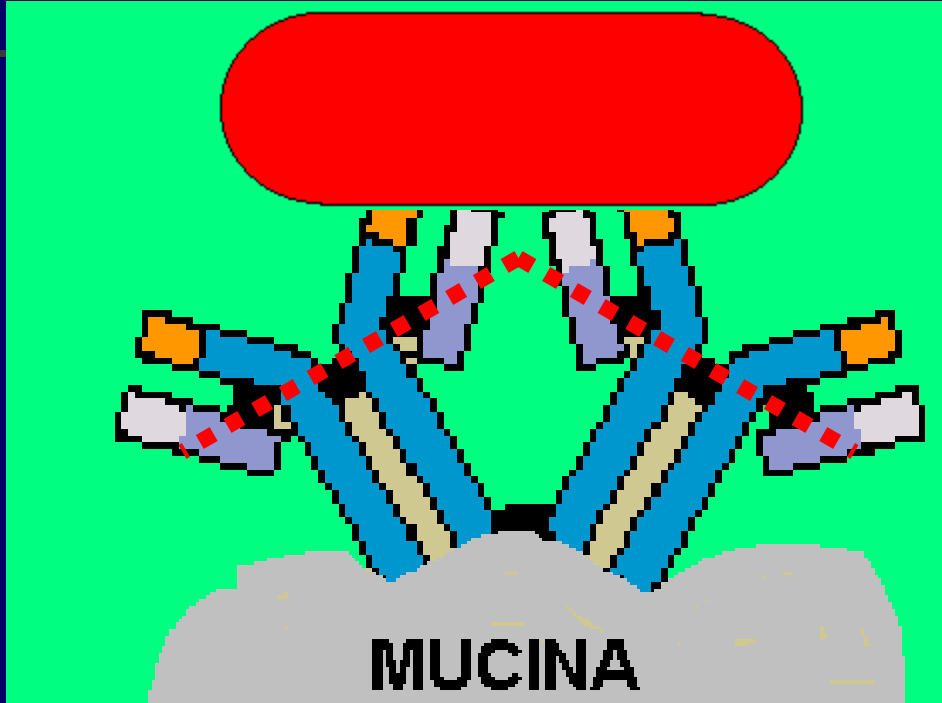
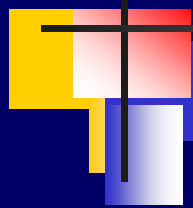
IgA1 hidrolasa



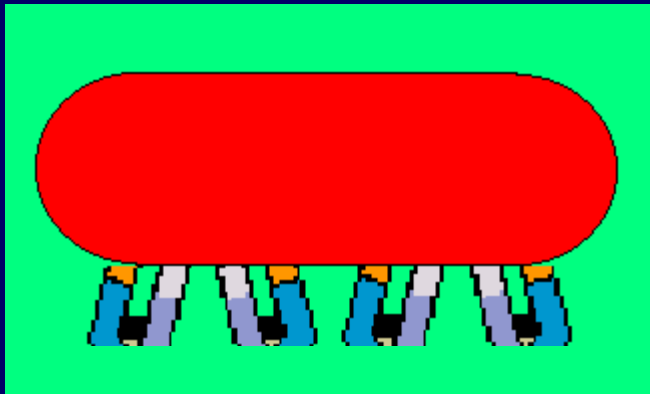
MUCINA

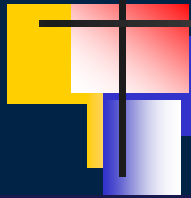




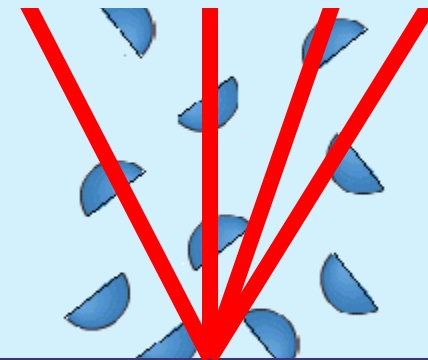
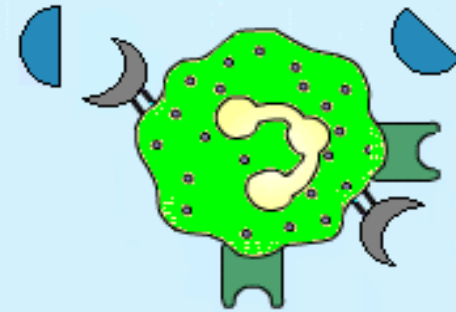
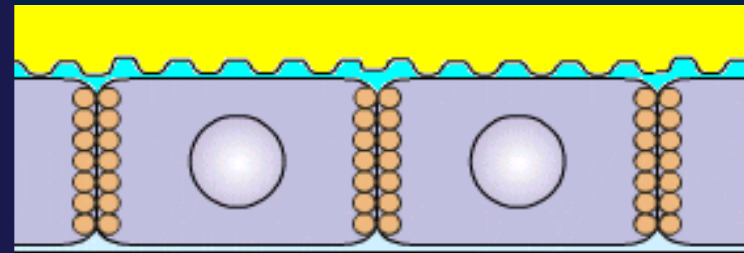
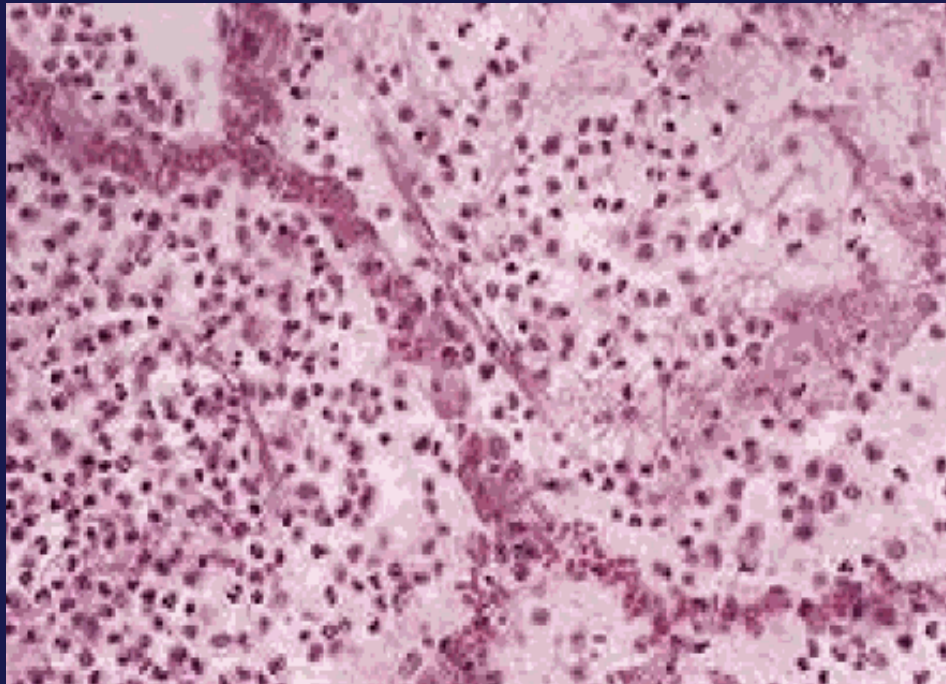


# IgA hidrolasas





# C5a peptidasa



*C5a peptidasa*



# **MOLÉCULAS QUE SUSTENTAN LA INVASIVIDAD BACTERIANA**

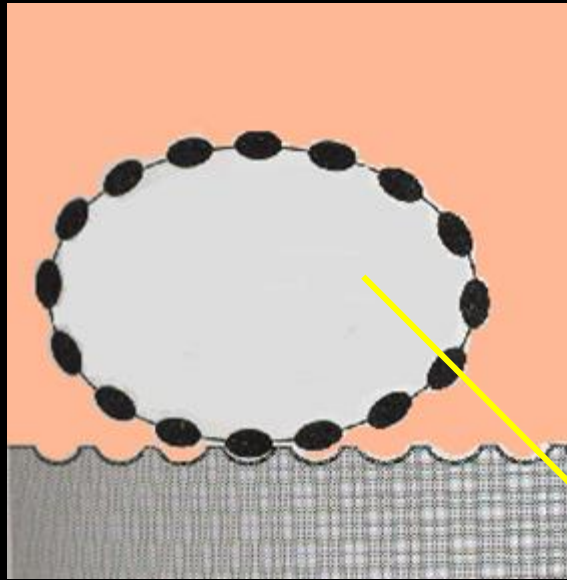
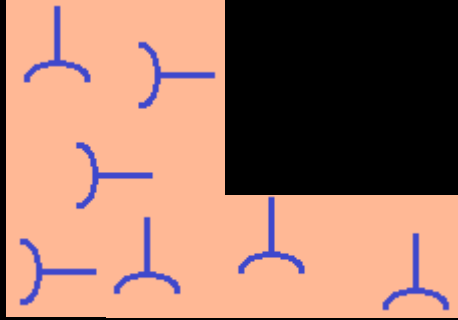
## **Reproducción-supervivencia**

### **4. Mecanismos de evasión de la defensa**

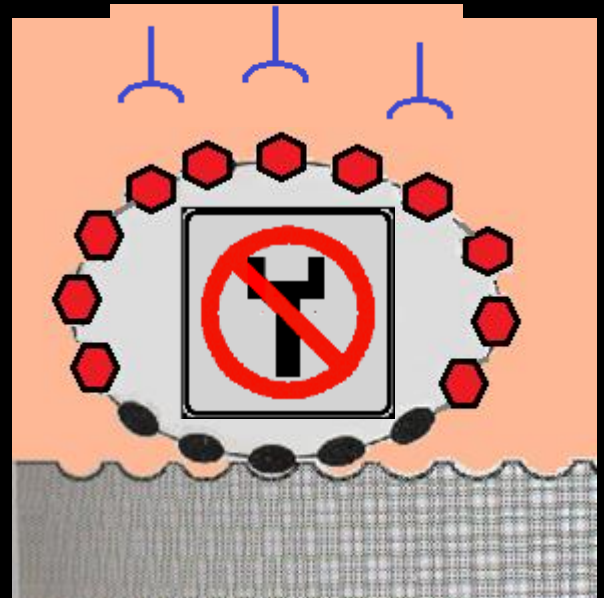
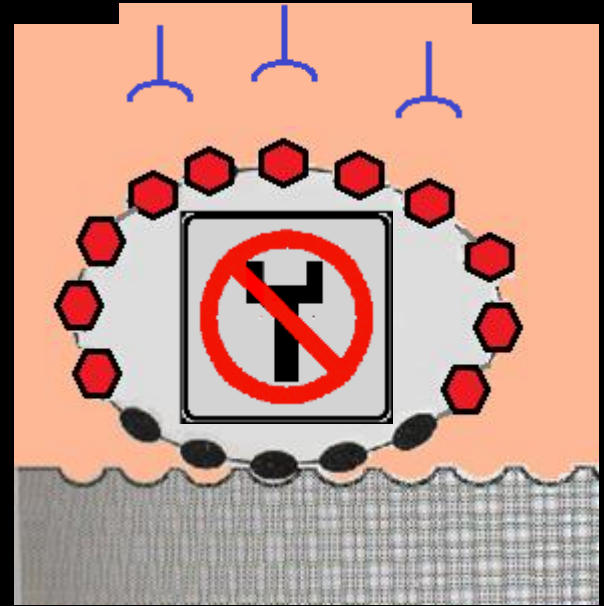
- ✓ **Variación antigénica**
- ✓ **Proteínas para camuflaje**
- ✓ **LPS: adsorción de ácido siálico y elongación**
- ✓ **Residencia intracelular**



# Variación antigénica



División

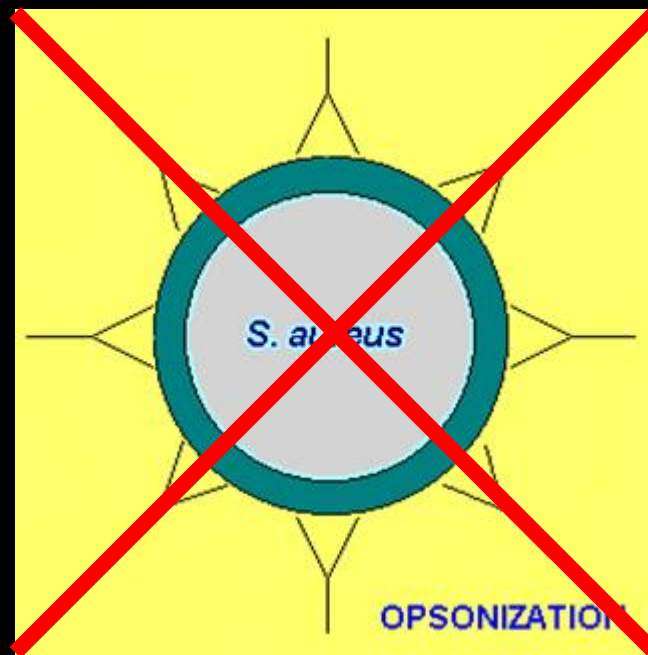
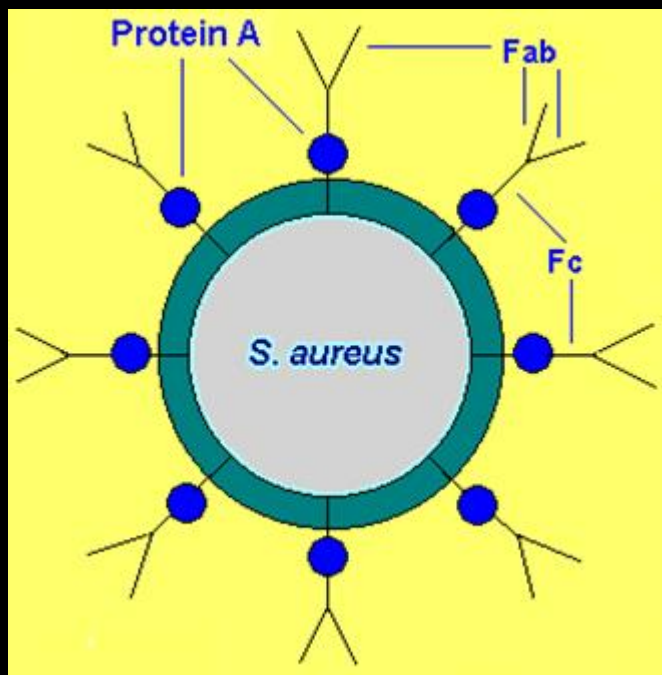
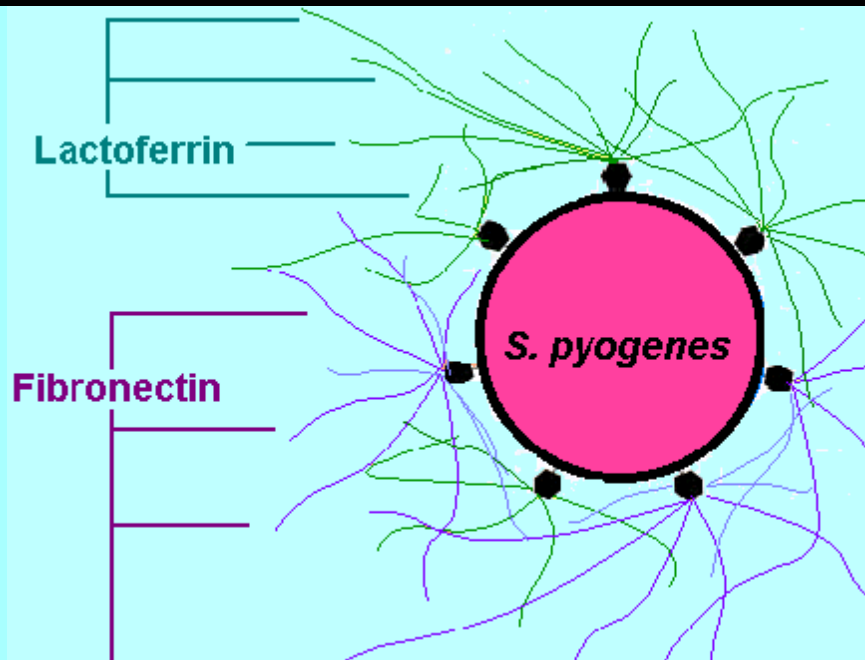
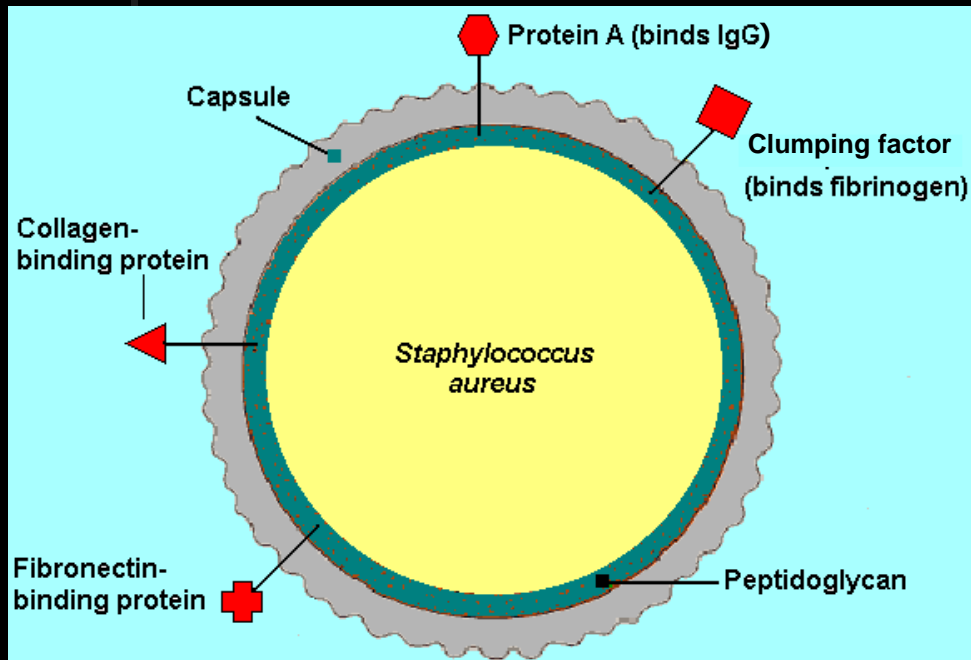




# Proteínas para camuflaje

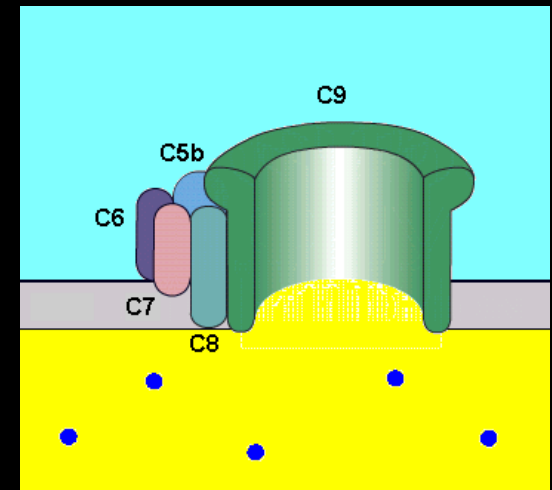
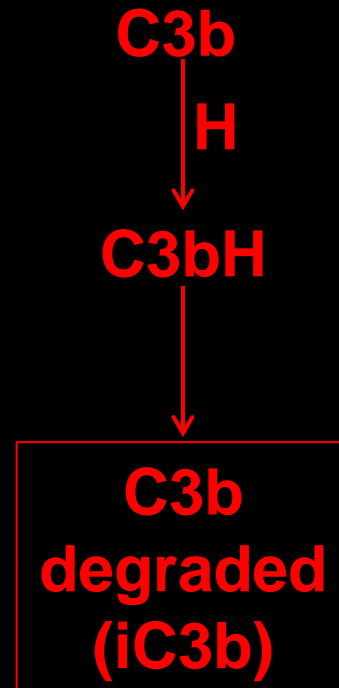
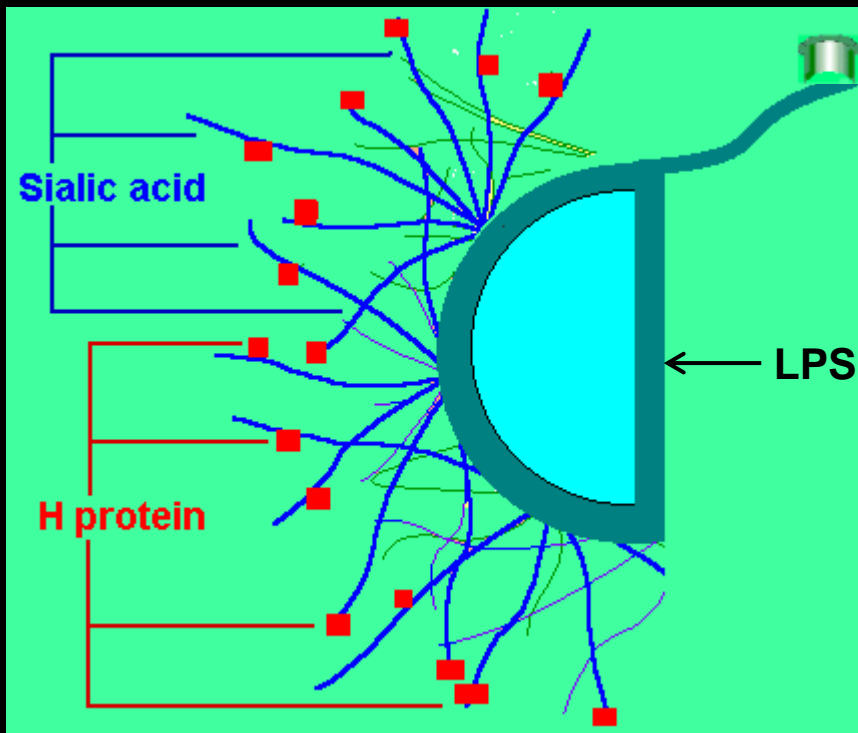
- **Proteína A: adsorbe Acs IgG por el Fc**
- **Proteína F: adsorbe fibronectina**
- **Sideróforos: adsorben lactoferrina, transferrina, etc.**





# LPS

- Adsorben ác. siálico hospedero, afín –a su vez – por la proteína H
- La elongación de sus cadenas neutraliza la acción del MAC







# MOLÉCULAS QUE SUSTENTAN LA INVASIVIDAD BACTERIANA

## Enzimas que inactivan antibióticos

**$\beta$ -lactamasas:** escinden el anillo  $\beta$ -lactámico (el ác. peniciloico no es reconocido por las PBPs)

**Aminoglucosidasas (acetil, fosforil y adenil transferasas)**

**Glucopetididasas:** las VanXHAB sustituyen con D-Ala-D-Lac al receptor D-Ala-D-Ala en el peptidoglicano

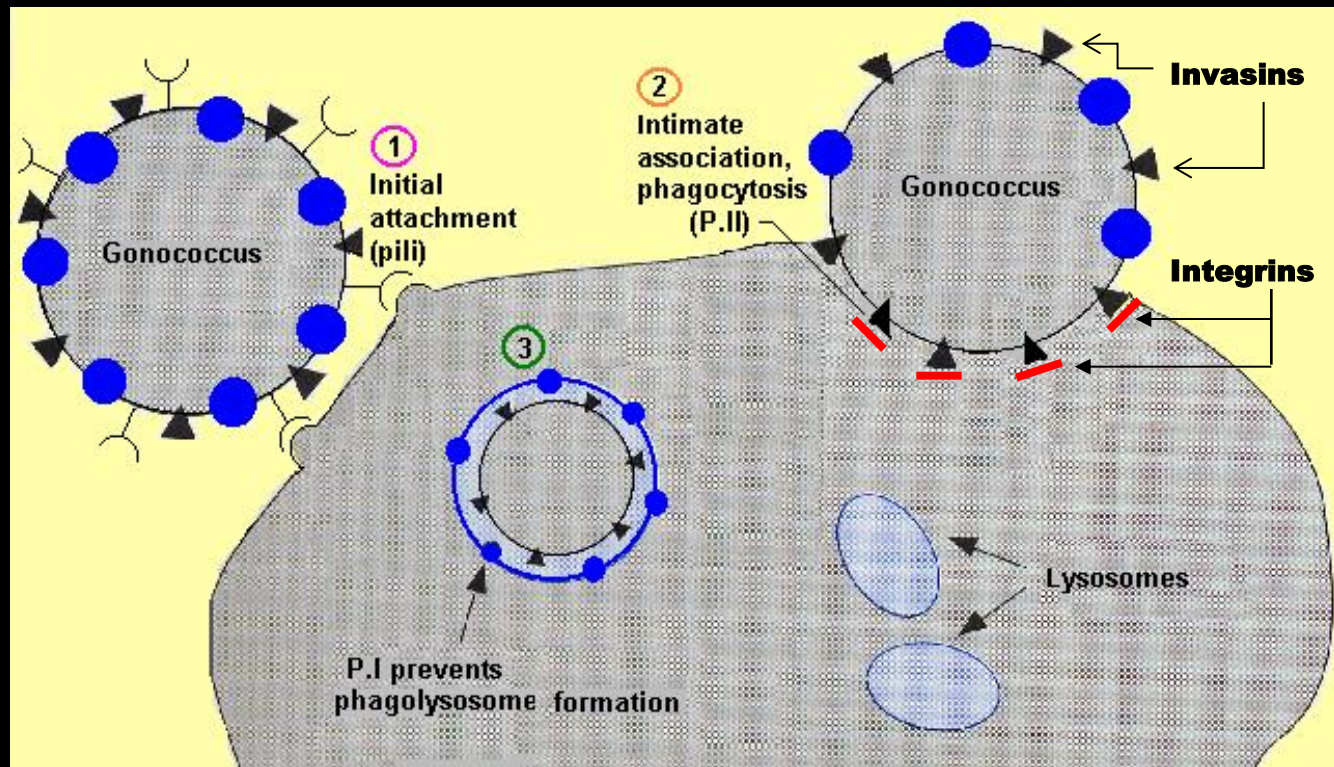
**Macrolidasas (Erm metila una adenina de la peptidil transferasa del rRNA 23S, receptora de la eritromicina)**

# MOLÉCULAS QUE SUSTENTAN LA INVASIVIDAD BACTERIANA

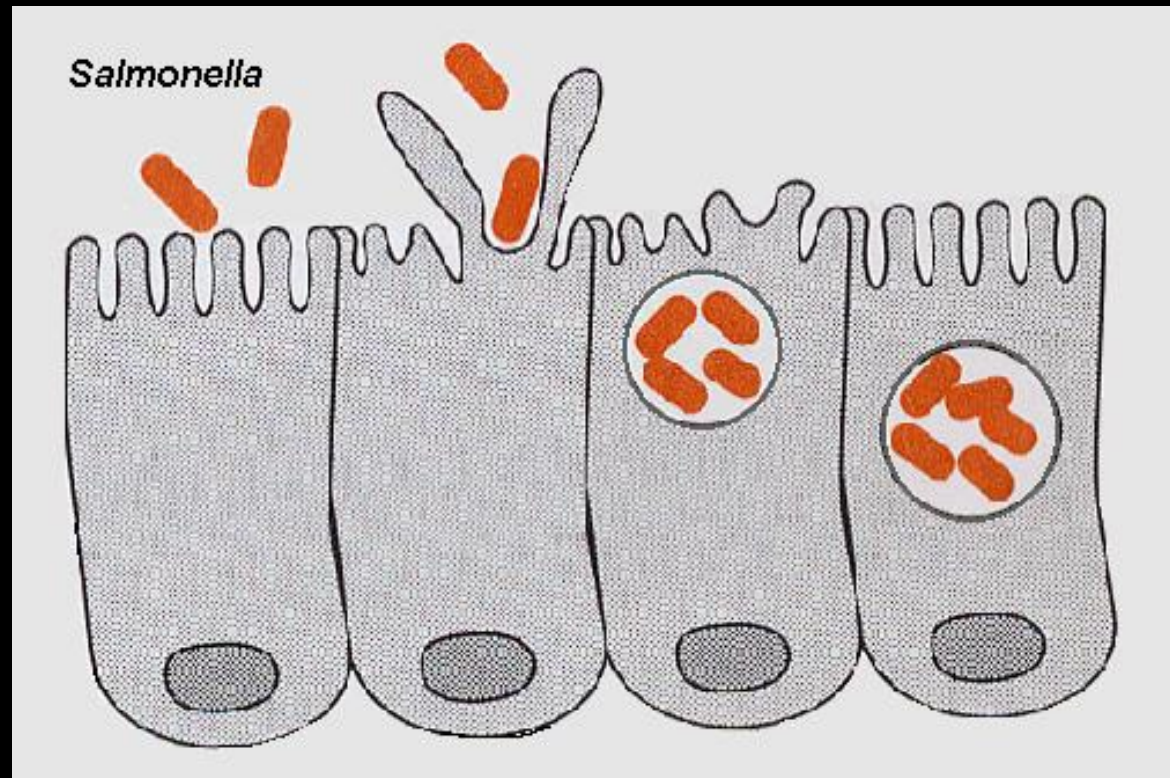
## Proteínas que inducen internalización

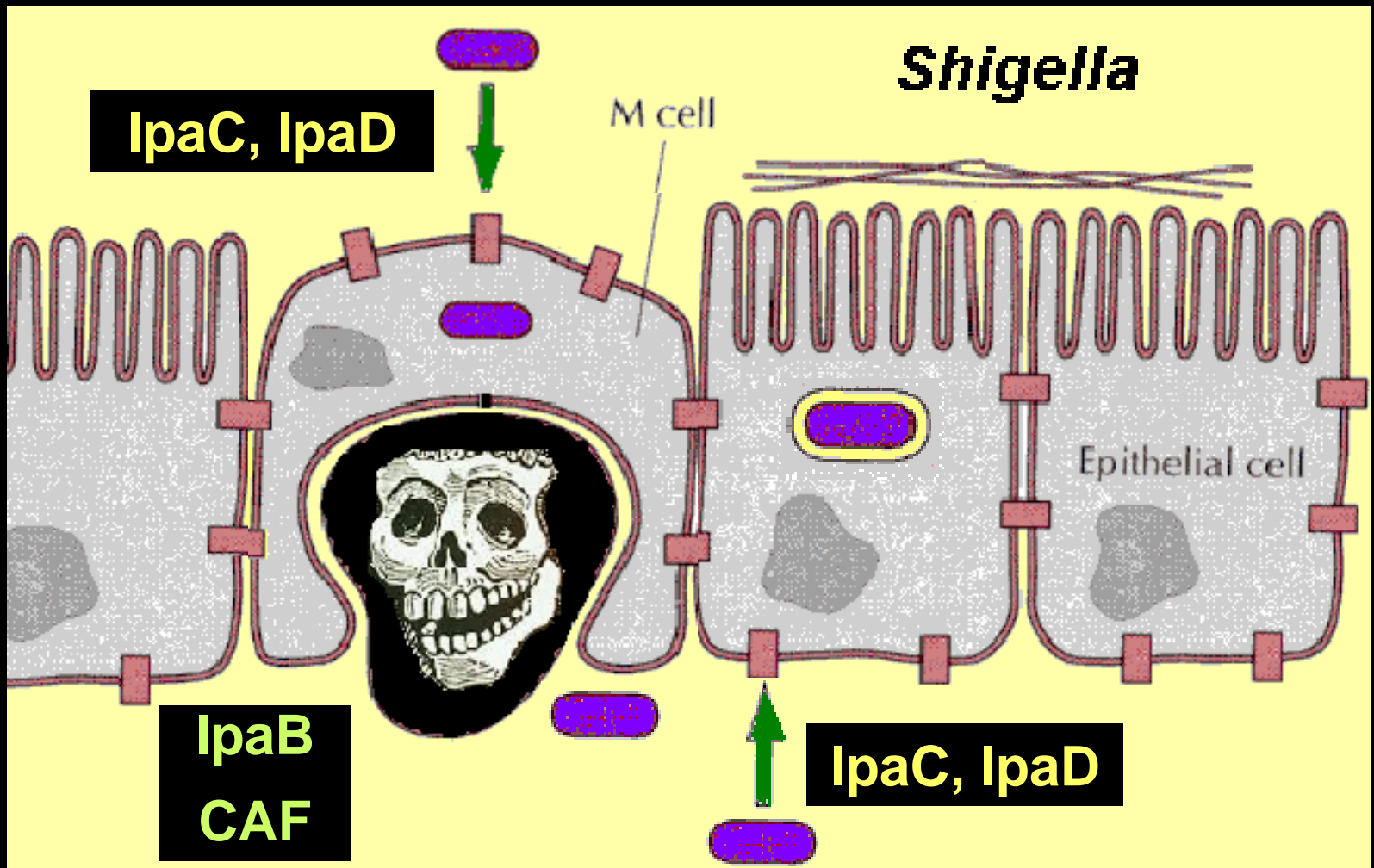
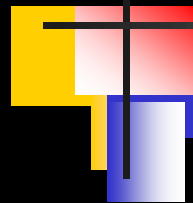
### Invasinas

Inducen la internalización bacteriana (residencia intracelular dentro de fagosomas o en el citosol)

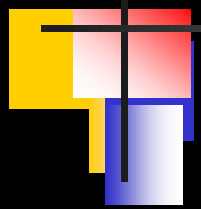


# Residencia intracelular







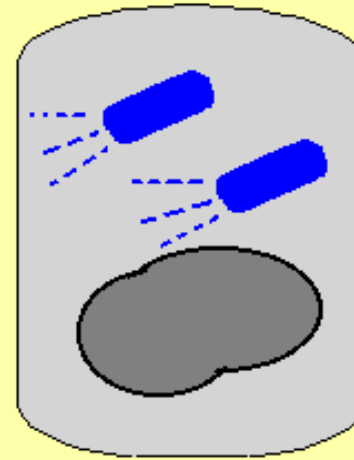
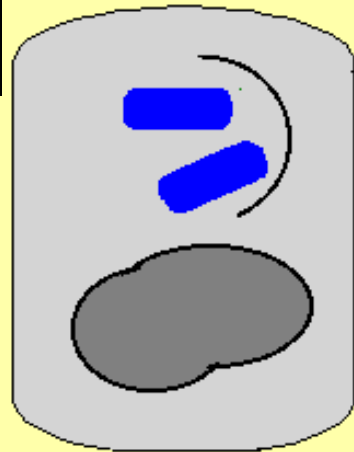


IpaB

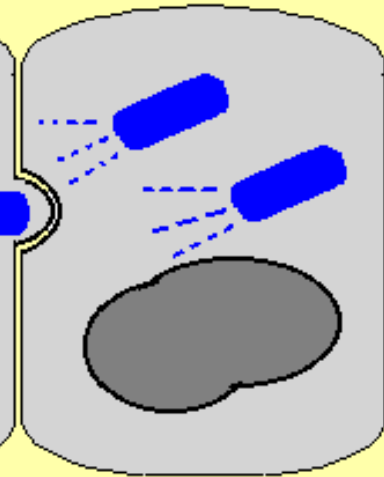
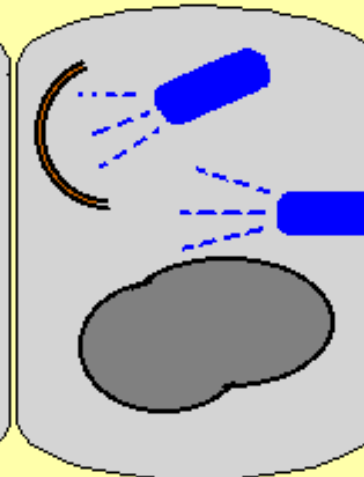
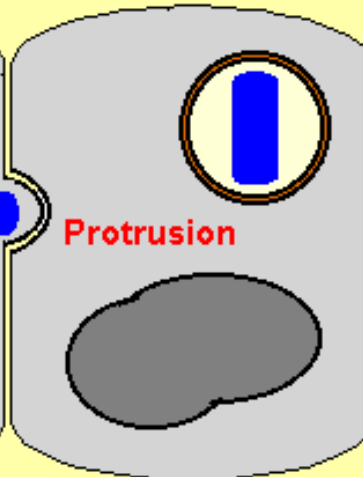
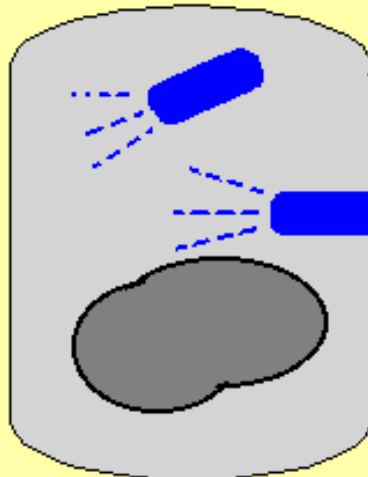
actin tails



Intracellular spread by *Shigella*



IpaB



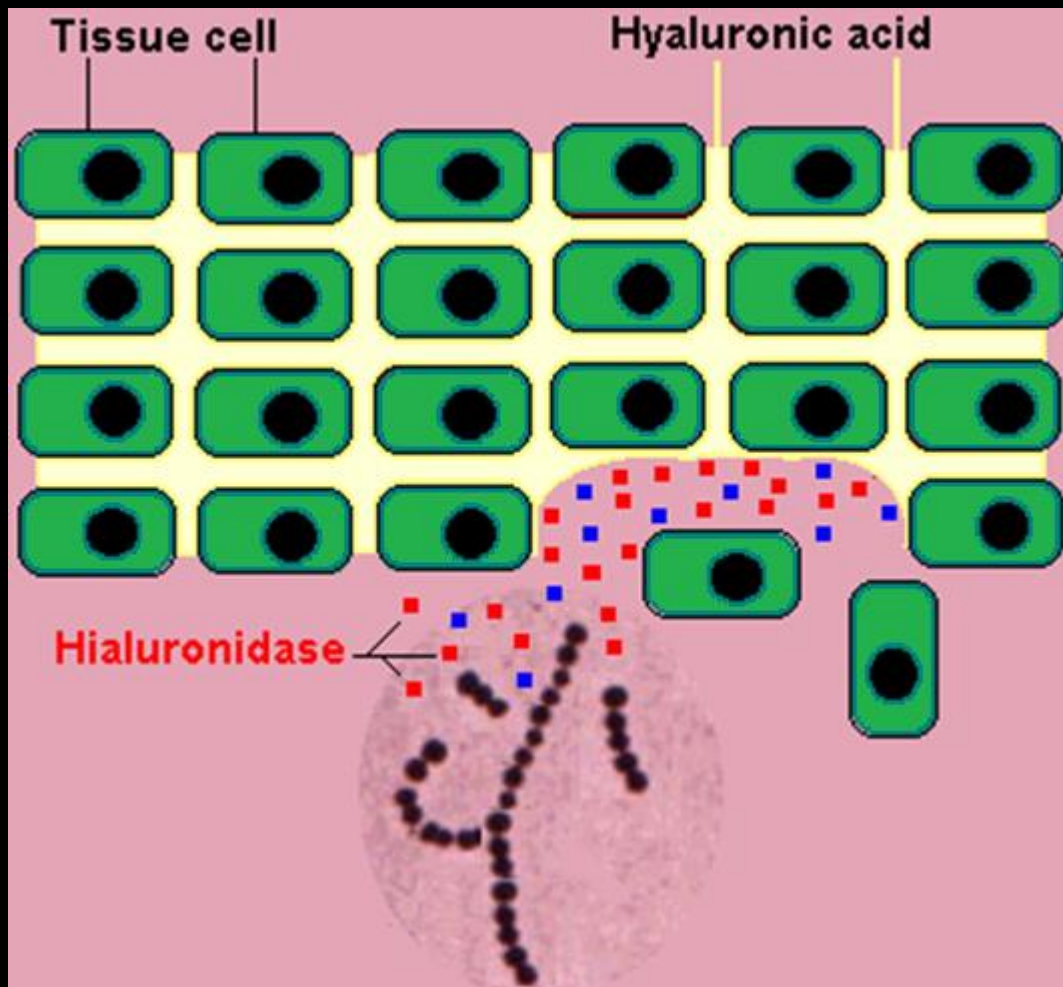
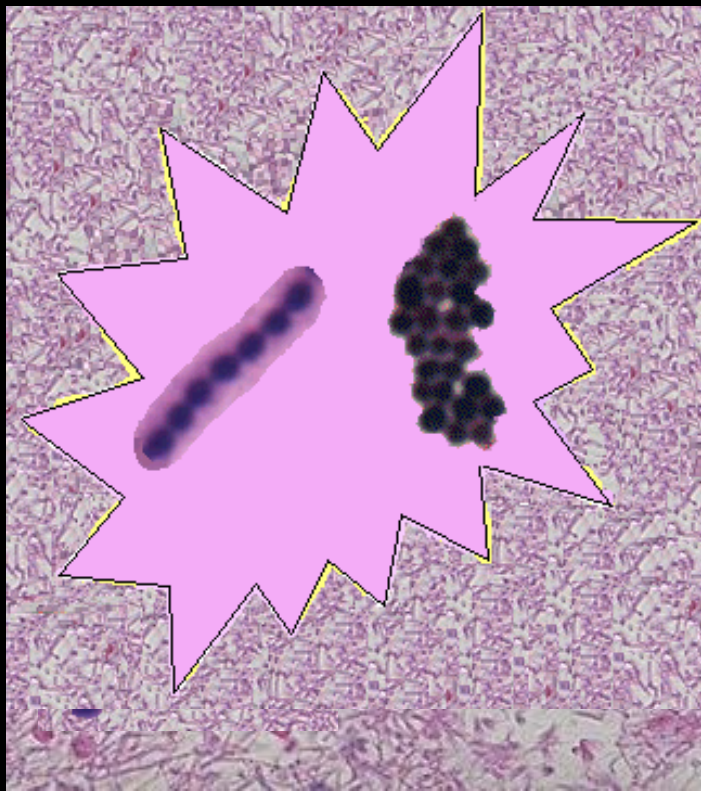
# MOLÉCULAS QUE SUSTENTAN LA INVASIVIDAD BACTERIANA

## Diseminación

Hialuronidasas

Colagenasas

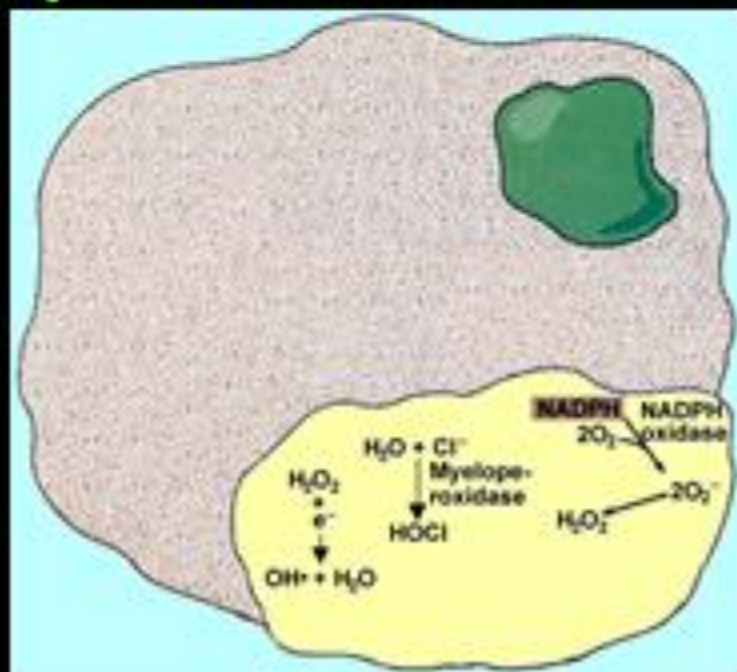
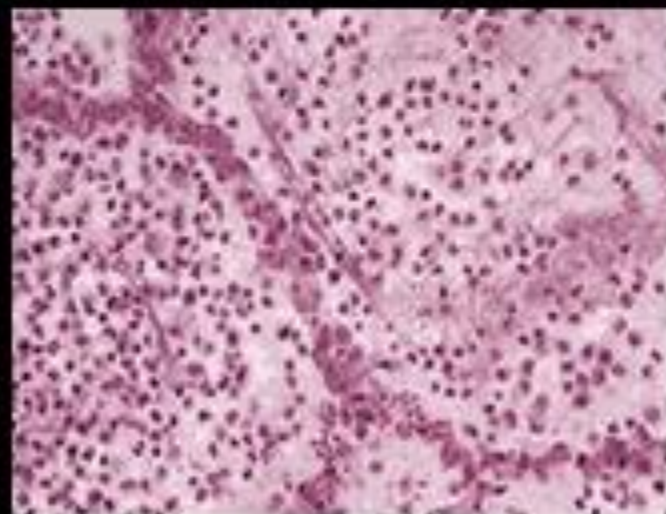
Fibrinolisin



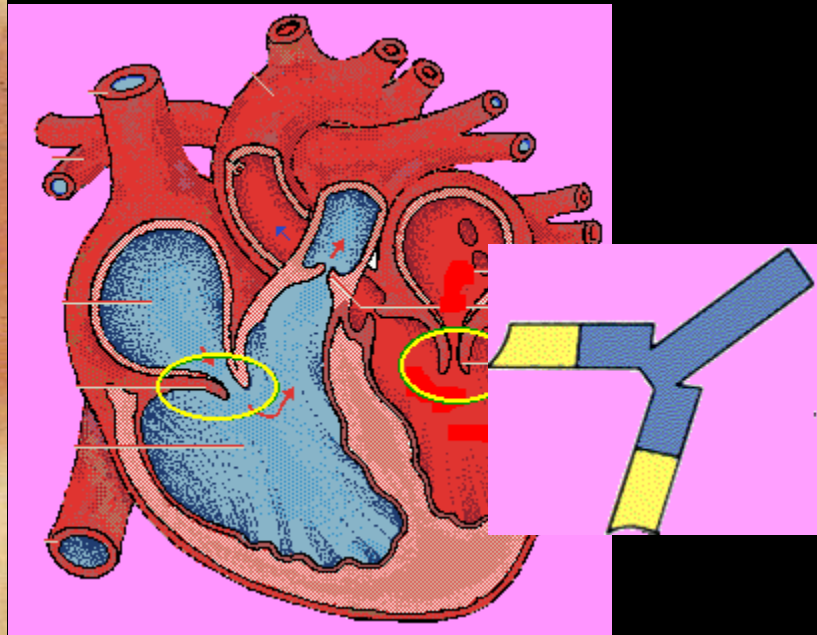
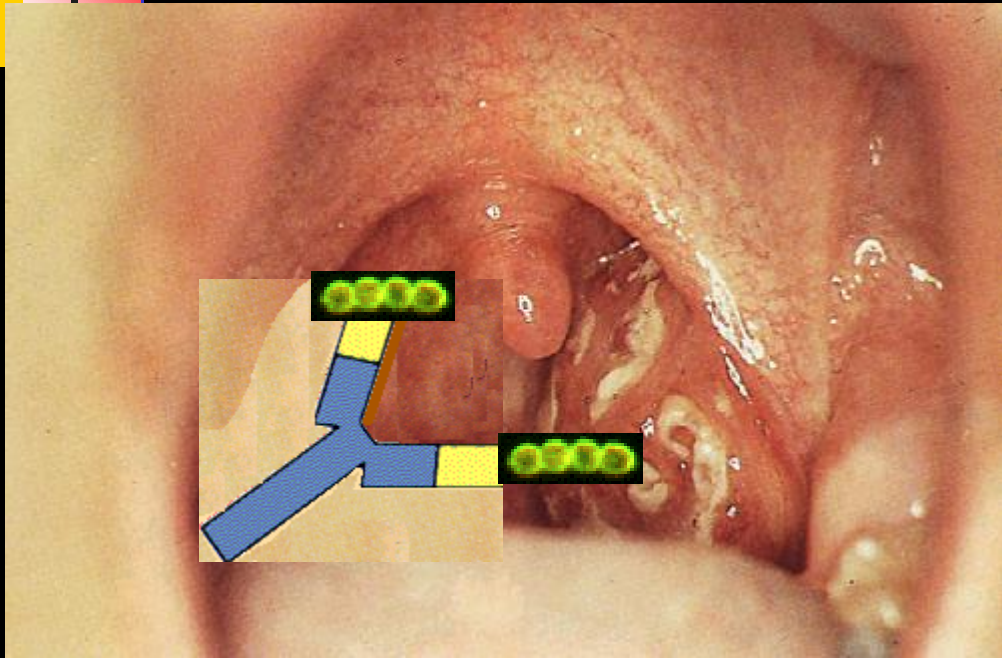
# BACTERIAS INVASIVAS EN LA PATOGENIA

**Desencadenan lesión "mayor", provocando:**

- Reacciones inflamatorias severas
- Procesos autoinmunes
- Reacciones de hipersensibilidad



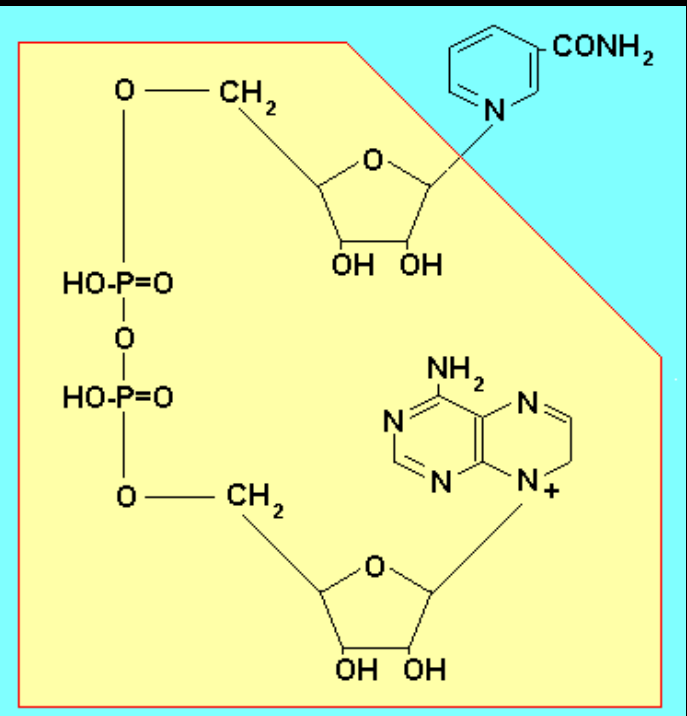
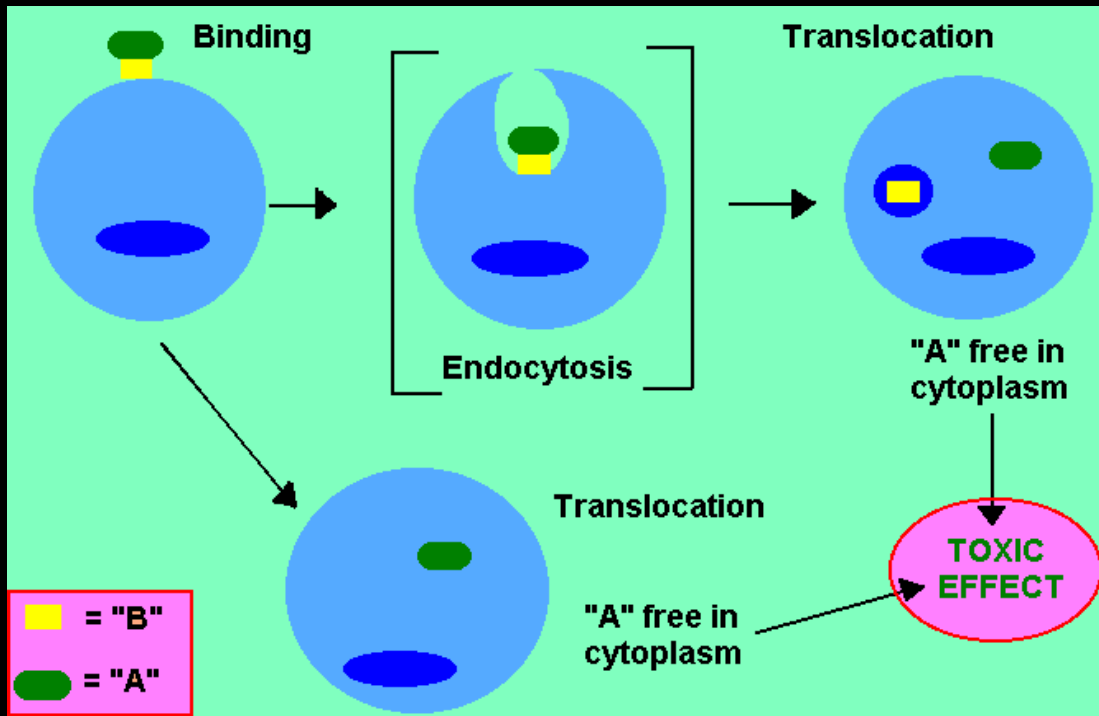




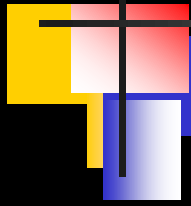


# EXOTOXINAS

✓ Del tipo A-B

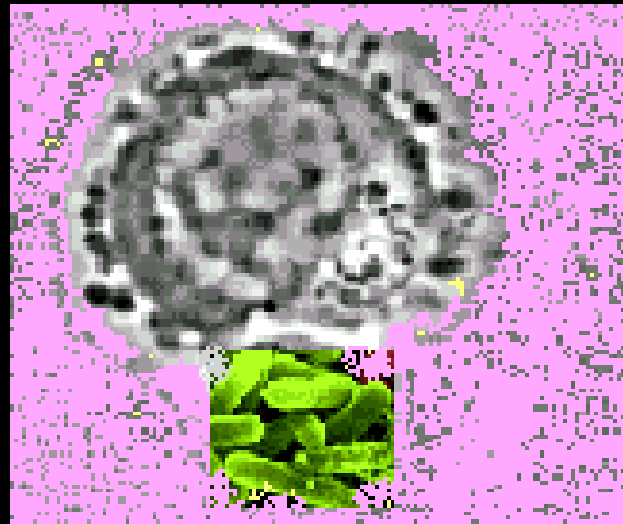


**ADP-ribosil**



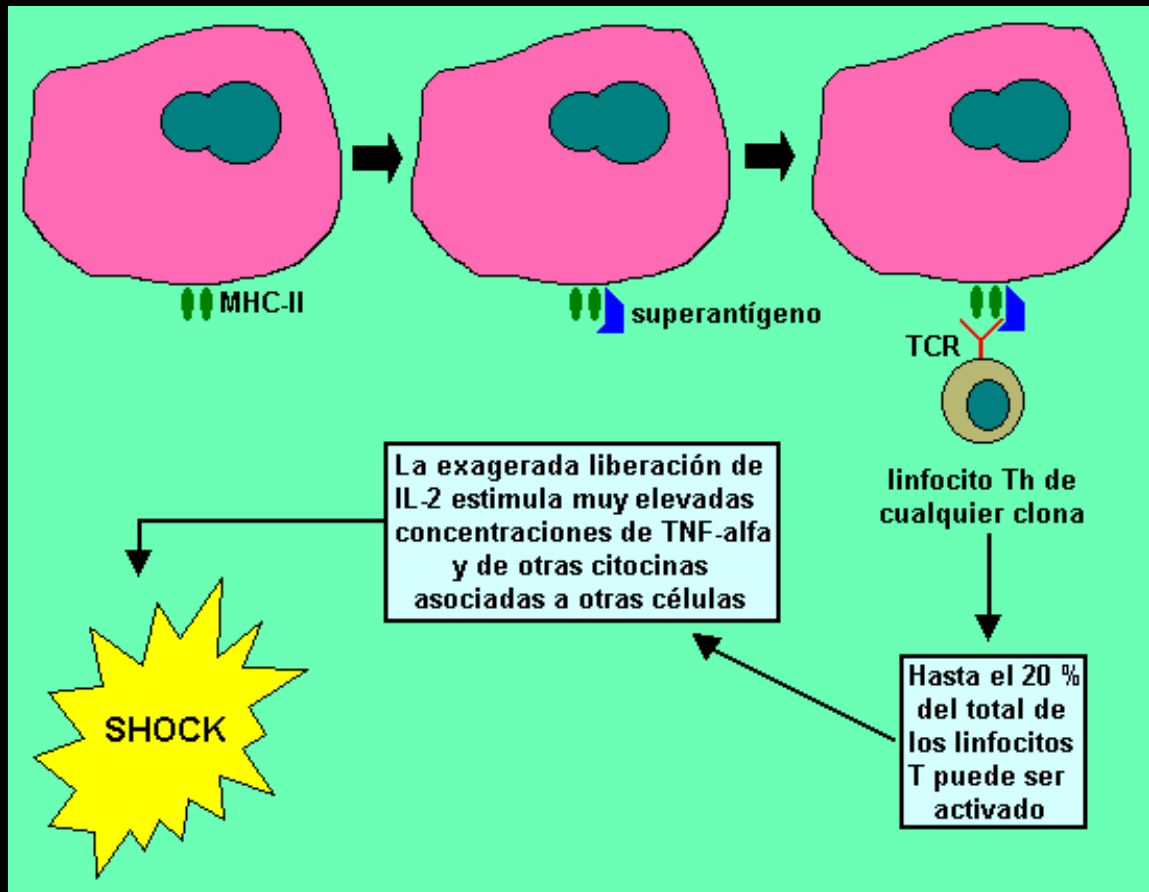
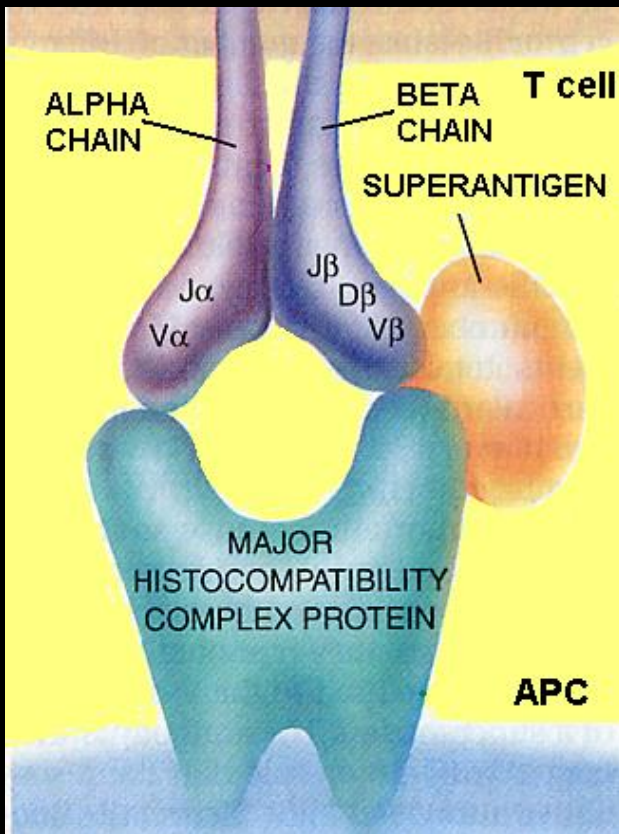
# EXOTOXINAS

- ✓ Que interrumpen la continuidad del sistema membranoso



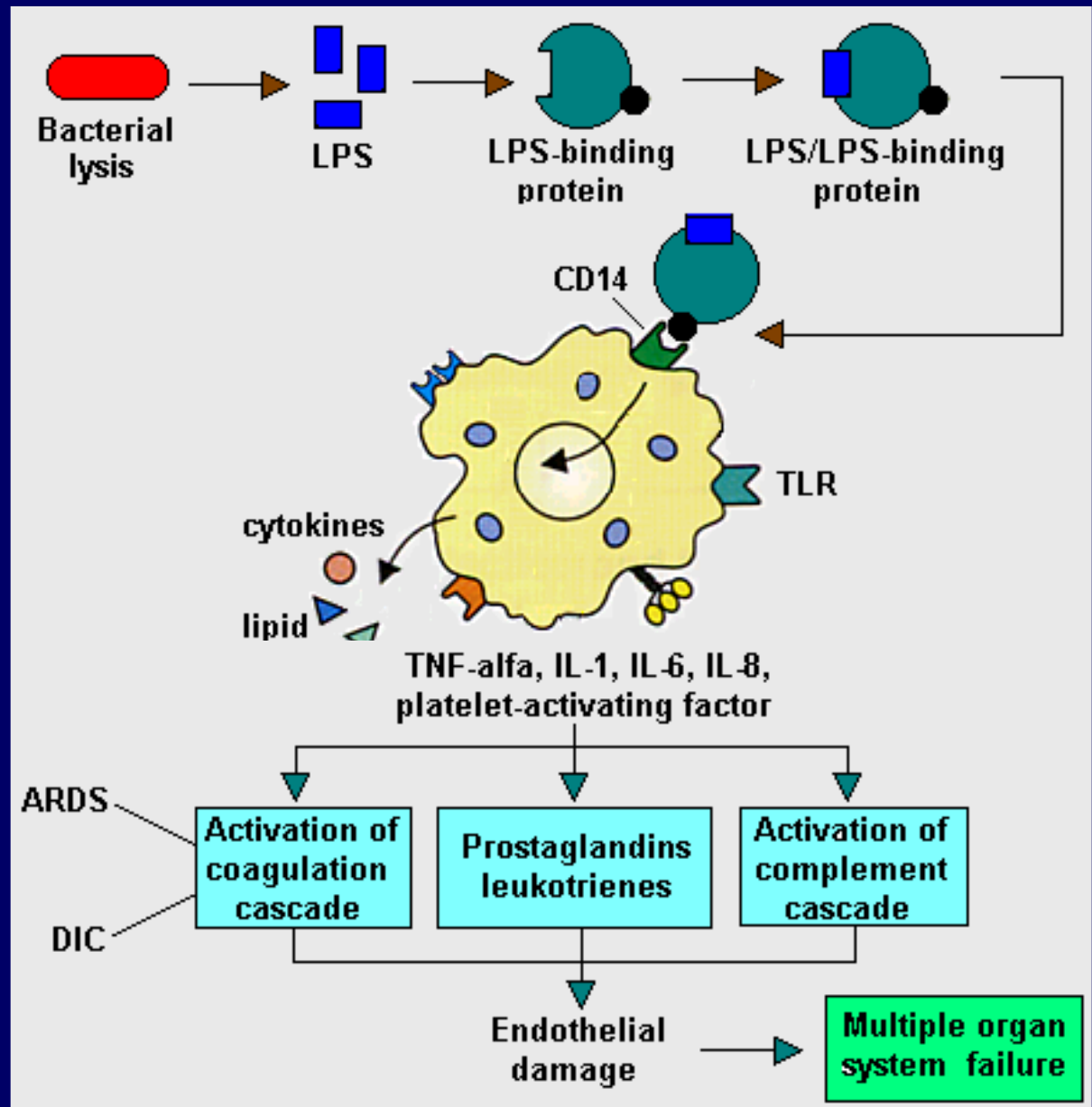
# EXOTOXINAS

✓ Que actúan como superantígenos



# ENDOTOXINAS (LPS, LOS)

- ✓ Lípido A (tóxico)
- ✓ Núcleo polisacarídico
- ✓ Polisacárido O (AgO)







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# Islas de patogenicidad

- **Conjunto de genes de virulencia agrupados en ciertos *locus* del cromosoma bacteriano**
- **Se sabe que proceden de otras clonas bacterianas (transferencia horizontal), porque su contenido G-C es diferente al del resto del cromosoma bacteriano**