Proteus, *Morganella* & *Providencia* species

These organisms primarily cause urinary tract infections, both community and hospital acquired

Learning objectives

- To know the special features, pathogenesis , clinical findings of diseases caused by Proteus, Enterobacter, Yersinia and Shigella
- To interpret the lab diagnosis of proteus, Enterobacter, Yersinia and Shigella

Proteus, Morganella & *Providencia* species

- All are normal intestinal flora
- Opportunistic pathogens
- All are non lactose fermentor

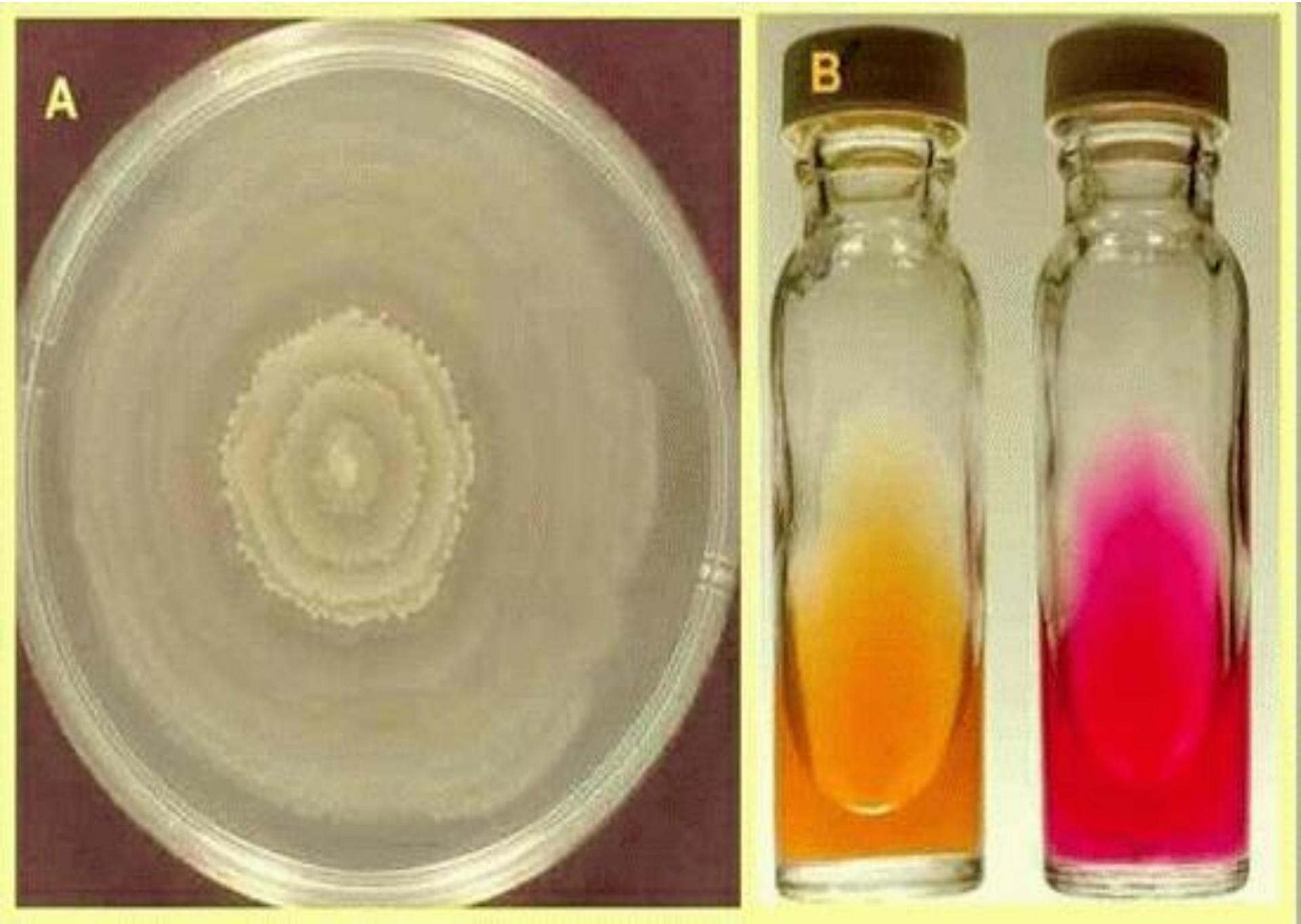
Proteus species

- *P. mirabilis* and *P. vulgaris* are widely recognized human pathogens
- Isolated from urine, wounds, and ear and bacteremic infections
- Very motile and produce striking **swarming** effect on blood agar, characterized by expanding rings (waves) of organisms over the surface of the agar.
- Both are **strongly urease positive**
- Both are phenylalanine deaminase positive

CULTURE CHARACTERISTICS

- Putrefactive (fishy) odor
- Swarming observed on NA, BA
- Pale colonies on MacConkey and DCA and no swarming

- Indole +
- H_2S +
- MR +, VP –
- Citrate +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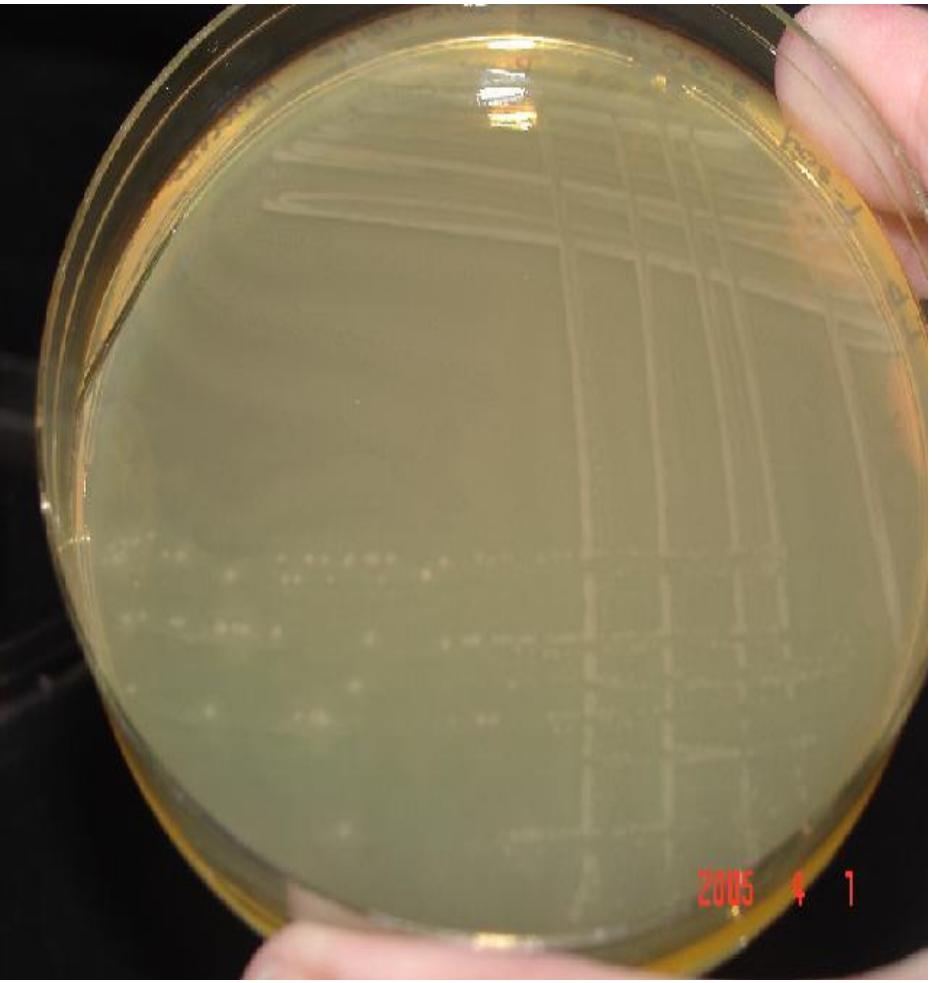
SWARMING OF PROTEUS ON NUTRIENT AGAR AND BLOOD AGAR



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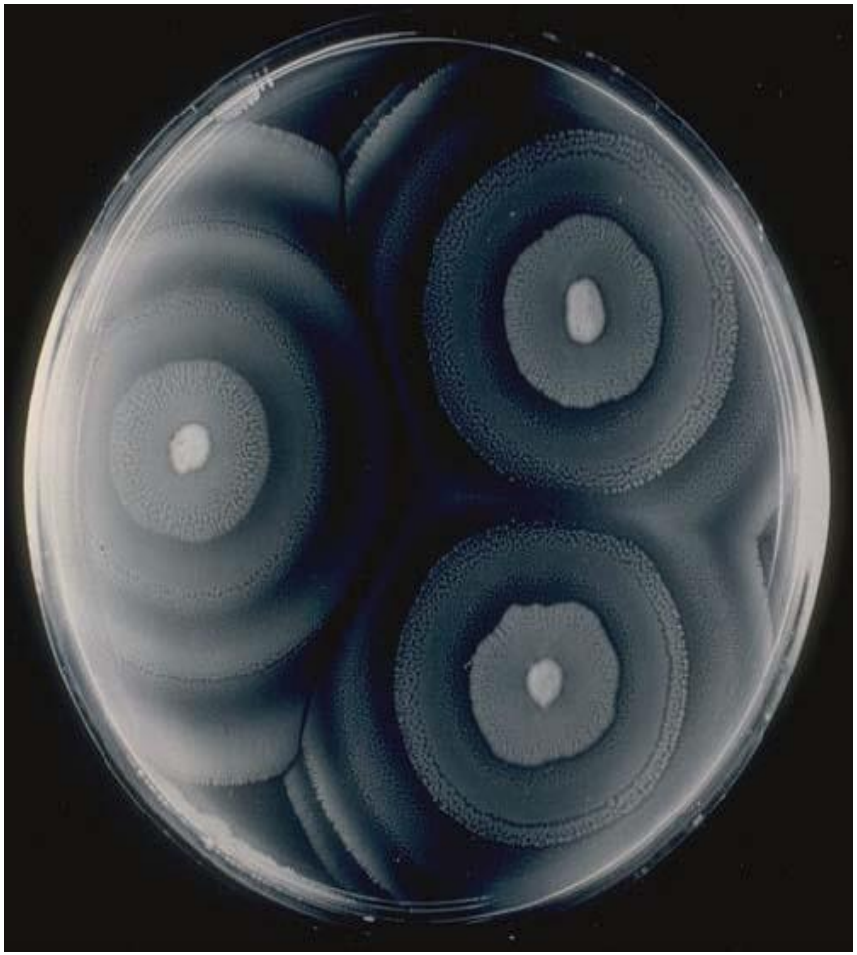
Swarming a Distinguishing Character in Proteus



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WEIL FELIX REACTION

- The cell wall O antigens of certain strains of *Proteus*, such as OX-2, OX-19 and OX-K, cross react with antigens of several species of rickettsiae. These *Proteus* antigens can be used in lab tests to detect the presence of antibodies against certain rickettsiae in patient serum. This test is called Weil-Felix reaction.

Pathogenesis

- Their tendency to cause UTI is probably due to their presence in the colon and to colonization of the urethra. The vigorous motility of *Proteus* organisms may contribute to their ability to invade the urinary tract.

Pathogenesis

- Production of enzyme urease is an important feature of this group. Urease hydrolyse the urea in urine to form ammonia, which raises the pH, producing alkaline urine. This encourages the formation of stones (calculi). Alkaline urine further encourage the growth of the organisms and extensive renal demage occurs.

TREATMENT

- Most strains are sensitive to aminoglycosides and septran but individual isolates can vary and frequently resistant to multiple antibiotics, sensitivity tests should be performed.

Enterobacter

Enterobacter is a genus of common Gram-negative, facultatively anaerobic, rod-shaped, non-spore-forming bacteria of the family Enterobacteriaceae.

Enterobacter

Several strains of these bacteria are pathogenic and cause opportunistic infections in immunocompromised (usually hospitalized) hosts and in those who are on mechanical ventilation.
The urinary and respiratory tracts are the most common sites of infection.

Enterobacter

The genus *Enterobacter* is a member of the coliform group of bacteria. It does not belong to the fecal coliforms group of bacteria, unlike *Escherichia coli*, because it is incapable of growth at **44.5 °C** in the presence of bile salts.

Biochemical characteristics

The genus *Enterobacter* ferments lactose with gas production during a 48-hour incubation at 35-37 °C in the presence of bile salts and detergents. It is oxidase-negative, indole-negative, and urease-variable.

Treatment

Treatment is dependent on local trends of antibiotic resistance.

- Cefepime.
- Imipenem (carbapenems) is often the antibiotic of choice.
- Aminoglycosides such as amikacin have been found to be very effective, as well.
- Quinolones can be an effective alternative.

Yersinia

Yersinia

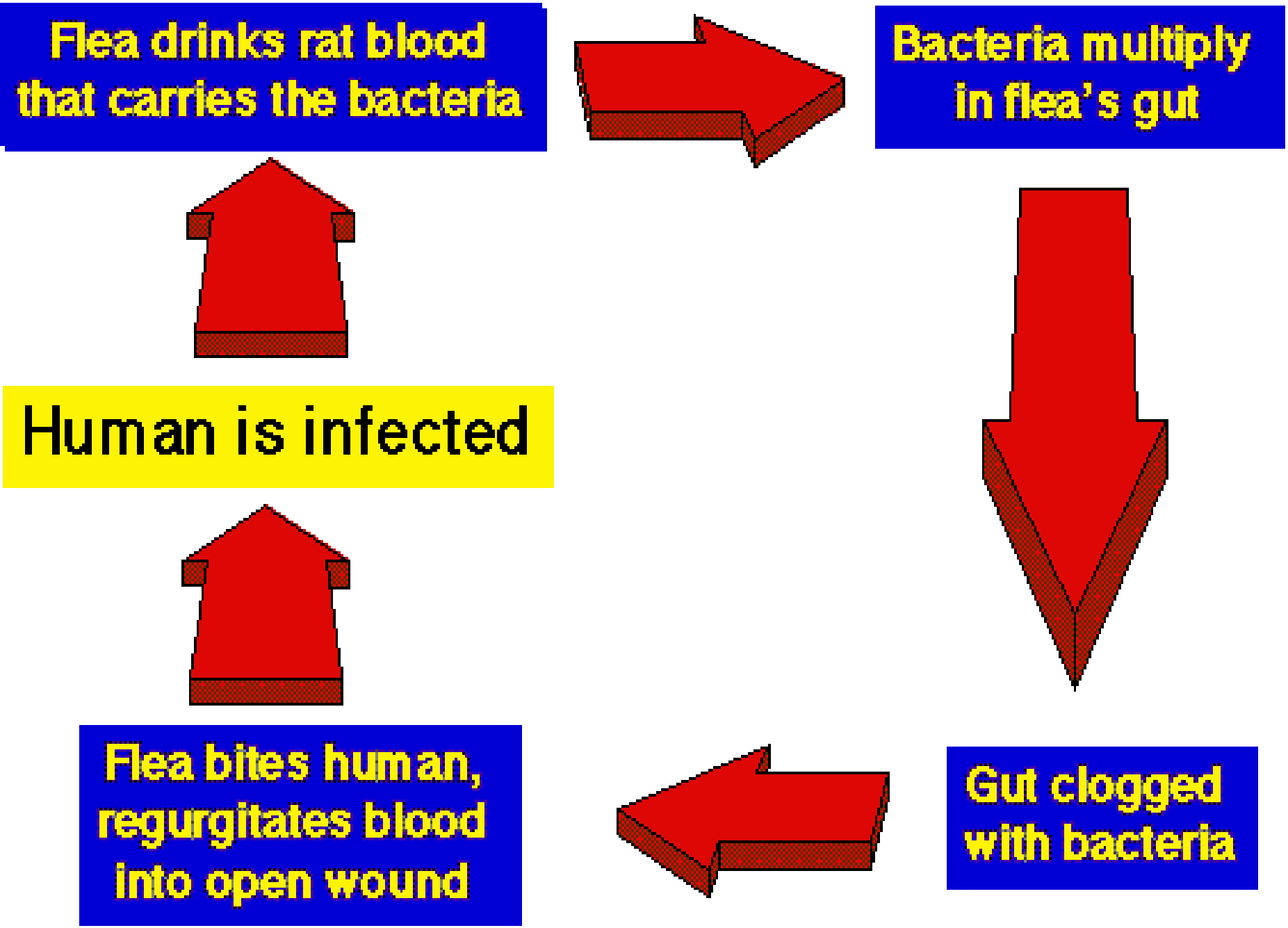
- Three species are important pathogens in man
- *Y. pestis*: plague ("black death")
- *Y. pseudotuberculosis* and *Y. enterocolitica*: gastroenteritis
  - Two forms of plague, bubonic and pneumonic
  - **Gram-negative, short, plump bacillus, exhibiting “safety-pin” or “bipolar” staining**

Yersinia

- **It is one of the most virulent bacteria known and has a strikingly low ID_{50} (i.e 1 to 10 organisms are capable of causing disease).**

Pathogenesis
Bubonic plague

- *Y. pestis* enters in flea when it feeds on an infected animal the bacteria multiply in the gut of the flea. A thick biofilm containing many organisms forms in upper GI Tract that prevent any food proceeding down. This blocked flea then regurgitates the organisms into the blood stream of the next animal or human it bites.


Flea drinks rat blood that carries the bacteria
Bacteria multiply in flea's gut
Human is infected
Flea bites human, regurgitates blood into open wound
Gut clogged with bacteria

Pathogenesis

Bubonic plague

- *Y. pestis* inoculated at the time of bite spread to the regional lymph nodes, which become swollen and tender. These swollen lymph nodes are the buboes that led to the name bubonic plaque. *Y. pestis* may reach the bloodstream and become widely disseminated. Hemorrhagic and necrotic lesions may develop in all organs and skin.

Pathogenesis

- **Primary pneumonic plague**

  Results from inhalation of infective droplets (usually from a coughing patient), with hemorrhagic consolidation of the lung, sepsis and death.

Buboes and pneumonia

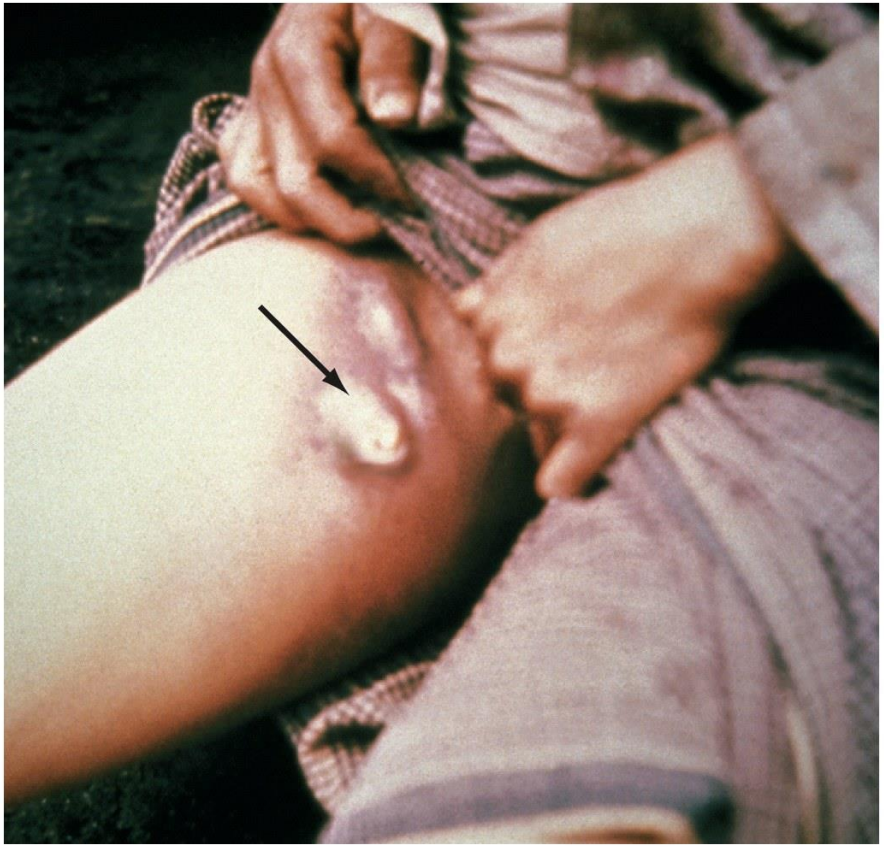


Figure 26.24a Microbiology: An Evolving Science
CDC

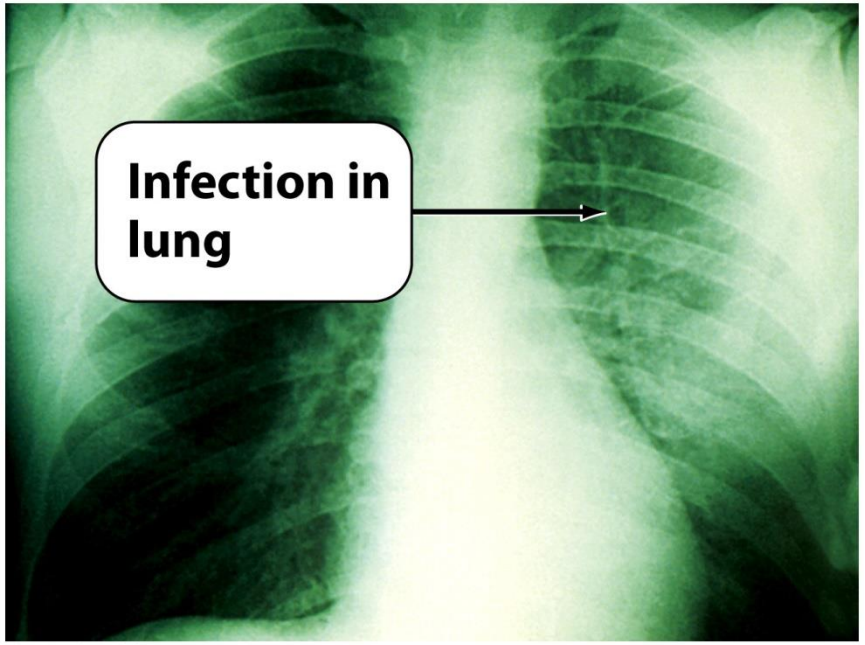


Figure 26.24d Microbiology: An Evolving Science
Science Source/Photo Researchers

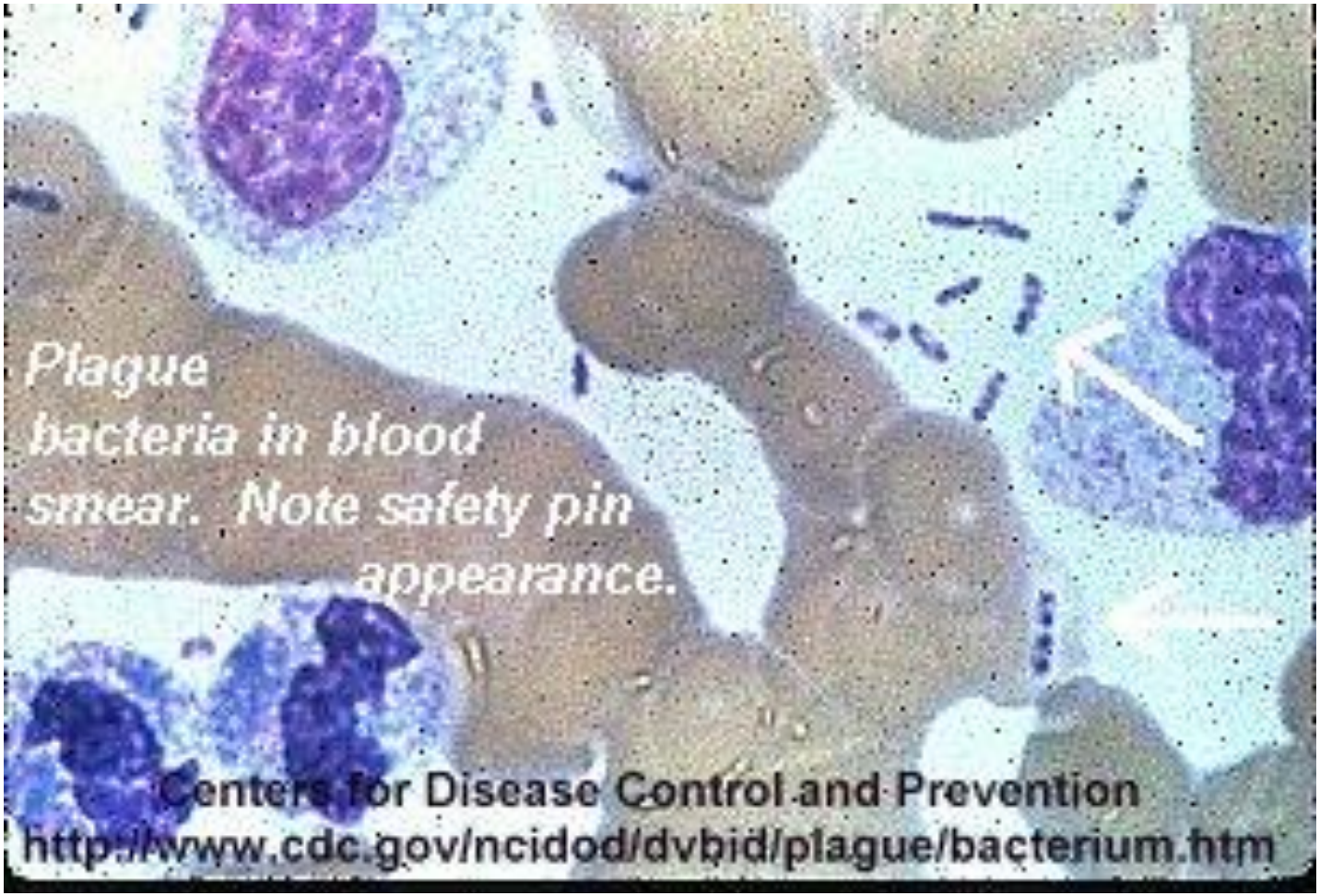
CLINICAL FINDINGS

- Bubonic plaque, which is the most frequent form, begins with pain and swelling of the lymph nodes draining the site of the flea bite and systemic symptoms such as high fever, myalgias and prostration. The effected nodes enlarge and become tender. Septic shock and pneumonia are the main life threatening subsequent events.

DIAGNOSIS

- Smear and culture of blood or pus from the bubo is the best diagnosis procedure. Giemsa or Wayson stain reveals the typical safety pin appearance of the organism.
- Fluorescent antibody staining.
- A rise in antibody titre to the antigen can be useful.

Yersinia pestis bipolar staining



Y. enterocolitica and *Y. pseudotuberculosis*

- They are found in the intestine of a variety of animals, and are transmissible to humans through contaminated food, drink or fomites, resulting in diarrhea, fever and abdominal pain that last for 1-2 weeks or, in some cases, months. Most are self-limited.
- *Y. enterocolitica* infection can cause pseudoappendicitis (enlarged mesenteric lymph nodes) in children

Y. enterocolitica and *Y. pseudotuberculosis*

- *Y. enterocolitica* infection can cause pseudoappendicitis (enlarged mesenteric lymph nodes) in children, and blood-transfusion related sepsis in those who used blood products stored for at least 4 weeks.

Y. enterocolitica and *Y. pseudotuberculosis*

– Identification

- *Y. pestis* can be separated from *Y. enterocolitica* and *Y. pseudotuberculosis* by the fact that it is non-motile.
- *Y. enterocolitica* and *Y. pseudotuberculosis* are both non-motile at 37^0 C, and motile at 22^0 C.
- *Y. pestis* is identified based on the following:
  - Non-motile
  - Bipolar staining
  - Slow growth of small colonies on ordinary culture media – it grows better at lower temperature (25-30^0 C)

TREATMENT

- The treatment of choice is combination of streptomycin and a tetracycline. Levofloxacin can also be use. There is no significant antibiotic resistance. Incision and drainage of the buboes are not necessary.

PREVENTION

- Prevention of plaque involves controlling the spread of rats in urban areas, avoiding both flea bites and contact with dead wild rodents. A patient must be placed in strict isolation(quarantine) for 72 hrs after antibiotic therapy is started.
- Reporting a case of plaque to the public health authorities is mandatory.

VACCINE

- Vaccine consisting of formalin killed organism provides partial protection against bubonic but not pneumonic plaque.

Shigella

Enterocolitis caused by Shigella is often called bacillary dysentery.

The term dysentery refers to bloody diarrhea.

Important Properties

- Non lactose fermenting gram negative rods.
- Shigella can be distinguished from salmonellae by three properties:

1) They produce no gas from the fermentation of glucose

2) They do not produce H_2S

3) Non-motile

Important Properties

- Shigella has four species that differ antigenically and, to a lesser extent, biochemically.
    - *S. dysenteriae* (Group A)
    - *S. flexneri* (Group B)
    - *S. boydii* (Group C)
    - *S. sonnei* (Group D)
    - All four species cause dysentery. Abdominal pain, Bloody stool, Diarrhea

Shigella

Pathogenesis

The organism is transmitted by the fecal oral route.

Shigellosis is primarily a pediatric disease, and is restricted to the GI tract. Children younger than 10 years account for approximately half of *Shigella* positive stool cultures

Some strains produce an enterotoxin called Shiga toxin. It is very similar to those produce by enterohemorrhagic *E. coli* O157:H7 strains that causes enterocolitis and HUS.

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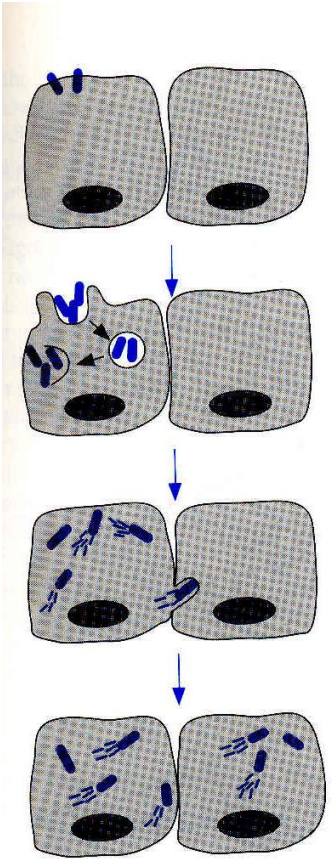
Shigella

Pathogenesis

Causes disease almost exclusively in the GI Tract, produce bloody diarrhea by invading the cells of the mucosa of colon and terminal ileum . Local inflammation accompanied by ulceration occurs, but the organisms rarely penetrate through the wall or enter the bloodstream, unlike salmonellae.

Shigella attachment and penetration



, Actin filaments; ●, Nucleus

Epidemiology

- *Shigellae* are the most effective pathogens among the enteric bacteria.
- They have a very low ID_{50}. Ingestion of as few as 100 organisms cause disease.
- Shigellosis is only a human disease (i.e., there is no animal reservoir).
- The four Fs—fingers, flies, food, and feces—are the principal factors in transmission.

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Epidemiology

- Outbreaks usually occur in day care nurseries and in mental hospitals.
- There is no prolong carrier state with *Shigella* infections, unlike seen in *S. typhi* infections.

Clinical Features

- Incubation period --- 1 to 4 days
- Symptoms begin with fever and abdominal cramps, followed by diarrhea, which may be watery at first but later contains blood and mucus.
- The disease varies from mild to severe depending upon two major factors the species and age of patient, young children and elderly are most severely effected.
- The diarrhea frequently resolves in 2 or 3 days; in severe cases, antibiotics can shorten the course.

Laboratory Diagnosis

- **Specimens**: fresh stool, mucus flecks, and rectal swabs. Large numbers of fecal leukocytes and some RBC may often be seen microscopically.
- **Culture**: differential and selective media as used for salmonellae.
- Non–lactose-fermenting (colorless) colonies on MacConkey's or EMB agar.

Non lactose fermenting colonies on MacConkey agar

Treatment

- The main treatment for shigellosis is fluid and electrolyte replacement.
- In mild cases, no antibiotics are indicated.
- In severe cases, a fluoroquinolone (e.g., ciprofloxacin) is the drug of choice.
- Trimethoprim- sulfamethoxazole is an alternative choice.
- Anti peristaltic drugs are contraindicated in shigellosis, because they prolong the fever, diarrhea, and excretion of the organism.

Prevention and control

- Humans are the only reservoir for shigellae.
- Prevention and control of dysentery:

  1) Sanitary control of water, food and milk; sewage disposal; and fly control.

  2) Isolation of patients and disinfection of excreta.

  3) Detection of subclinical cases.

There is no vaccine