



Q fever and other reckettsia

**Dr.Eman Albataineh,
Prof. Immunology
College of Medicine, Mu'tah university
Hemo. Module, 3rd year medical students**

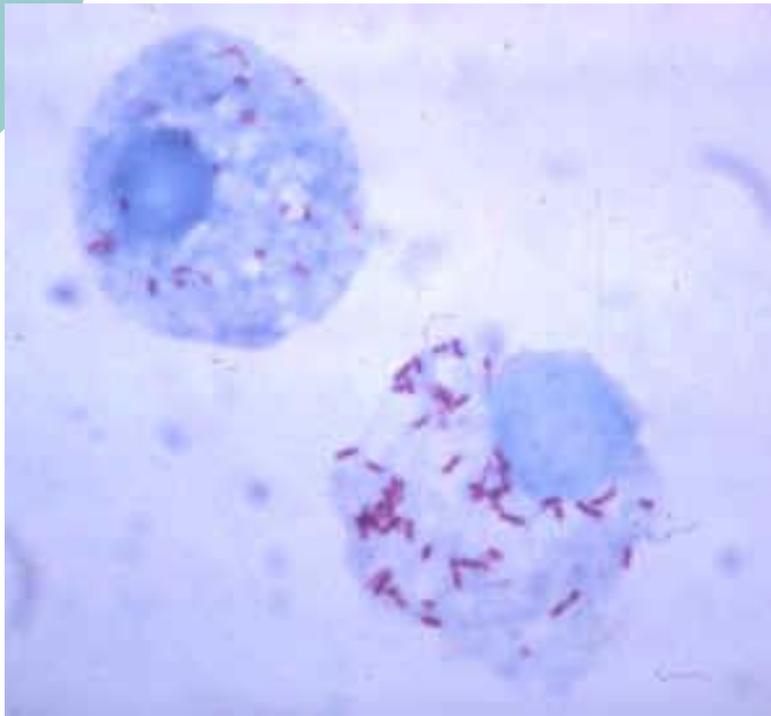
Category of rickettsia

- Species GROUPS
- **1-TYPHUS**
 - *Rickettsia prowazekii* (epidemic typhus),
 - *Rickettsia typhi* (endemic typhus),
- **2-SPOTTED FEVER GROUP**
 - *Rickettsia rickettsii* (spotted fever),
 - *Rickettsia conorii* (Mediterranean spotted fever)
 - *R. akari* (Rickettsial pox)
- **3-trench fever group**
 - *bartonella* (trench fever),
- **4- scrub typhus group**
 - *R. tsutsugamushi*

Bacteriology

- Small, pleomorphic cocco, rods or thread like
 - Gram stain poorly, but appear to be G-
 - Intracellular parasites
 - Stain readily with Giemsa
 - Resemble viruses in that they are obligate intracellular
 - Contain both DNA and RNA
 - All are transmitted by arthropod vectors as mite, ticks and lice
- **Arthropods, serves as both vector and reservoir**

Biological features



- Microcapsule and slim layer
- Culture : in yolk sacs of embryo eggs
- Cultivation is costly and hazardous because aerosol transmission can easily occur

Microbiology and General Characteristics

- Have cell wall, bigger than virus but smaller than bacteria
- Have an ATP transport system that allows them to use host ATP
- Sensitive to antibiotics (tetracycline)

R. antigens

- 1-shared with proteus strains as OX19 and OX2. is the basis for Weil-Felix reaction
- Group specific soluble antigens
- Strain specific antigens



Transmission

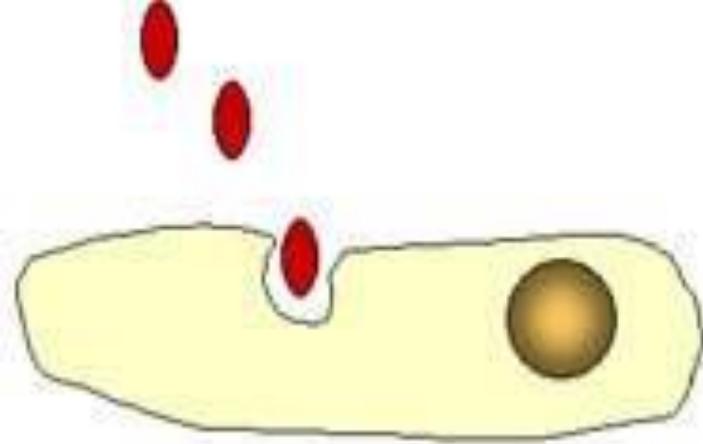
- spotted fever group and trench fever are transmitted via arthropod vectors taking the disease from rodents;
- Typhus fever group transmitted via arthropod vectors taking the disease from man

pathogenesis

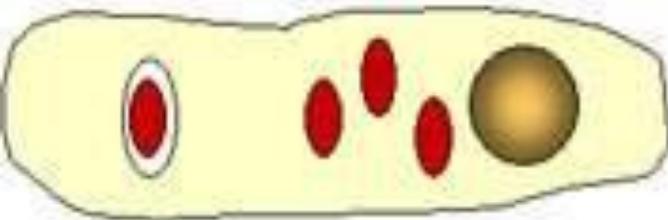
- Rickettsiae microorganisms appear to exert their pathologic effects by adhering to and then invading the endothelial lining of the vasculature within the various organs affected causing **heamorrhage** for that there is a red papule develop at site of mite bite.
- **Cell to cell transmission**; Once inside cell, the rickettsial organisms either multiply and accumulate in large numbers before lysing the host cell (typhus group) or they escape from the cell, damaging its membrane (filopodium local lysis)and causing the influx of water (spotted fever group) or they form pudding on membrane as in scrup typhus

- 
-
- Rickettsiae rely on the cytosol of the host cells for growth. To **avoid phagocytosis** within the cells, they secrete phospholipase D and hemolysin C, which disrupt the phagosomal membrane, allowing for rapid escape.
 - The most important pathophysiologic effect is **increased vascular permeability** with consequent edema, loss of blood volume, hypoalbuminemia, decreased osmotic pressure, and hypotension

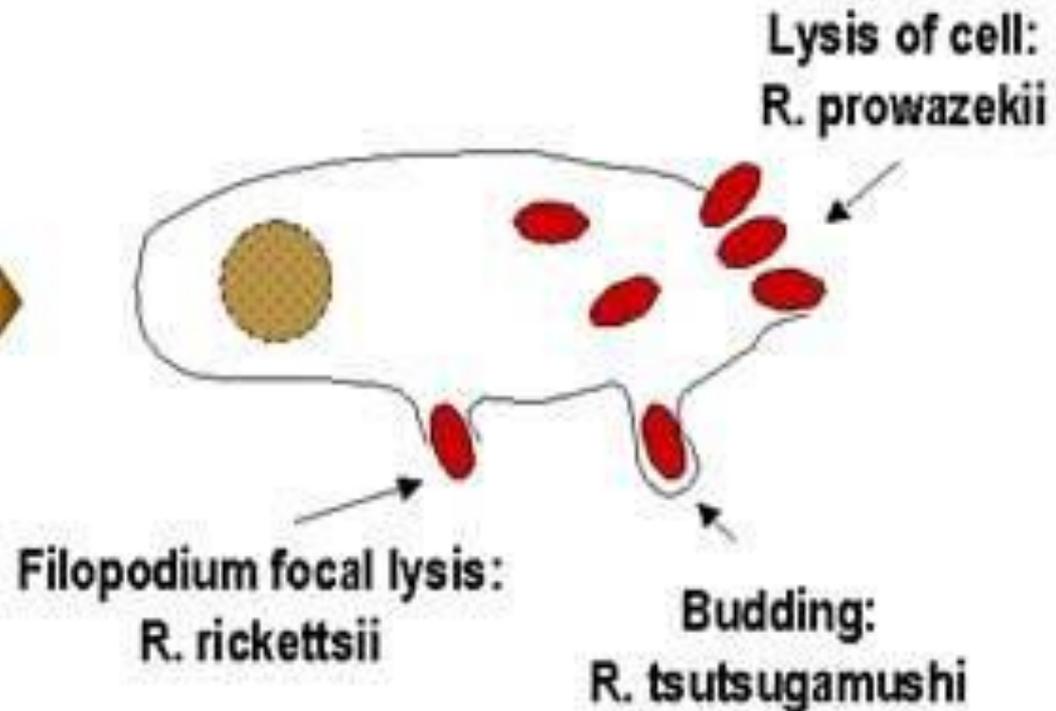
Cell to cell transmission



Rickettsia infection of
an endothelial cell
Phagocytosis is induced



Bacterium escapes
from phagosome



Rickettsia cell-to-cell spread

Attachment and phagocytosis

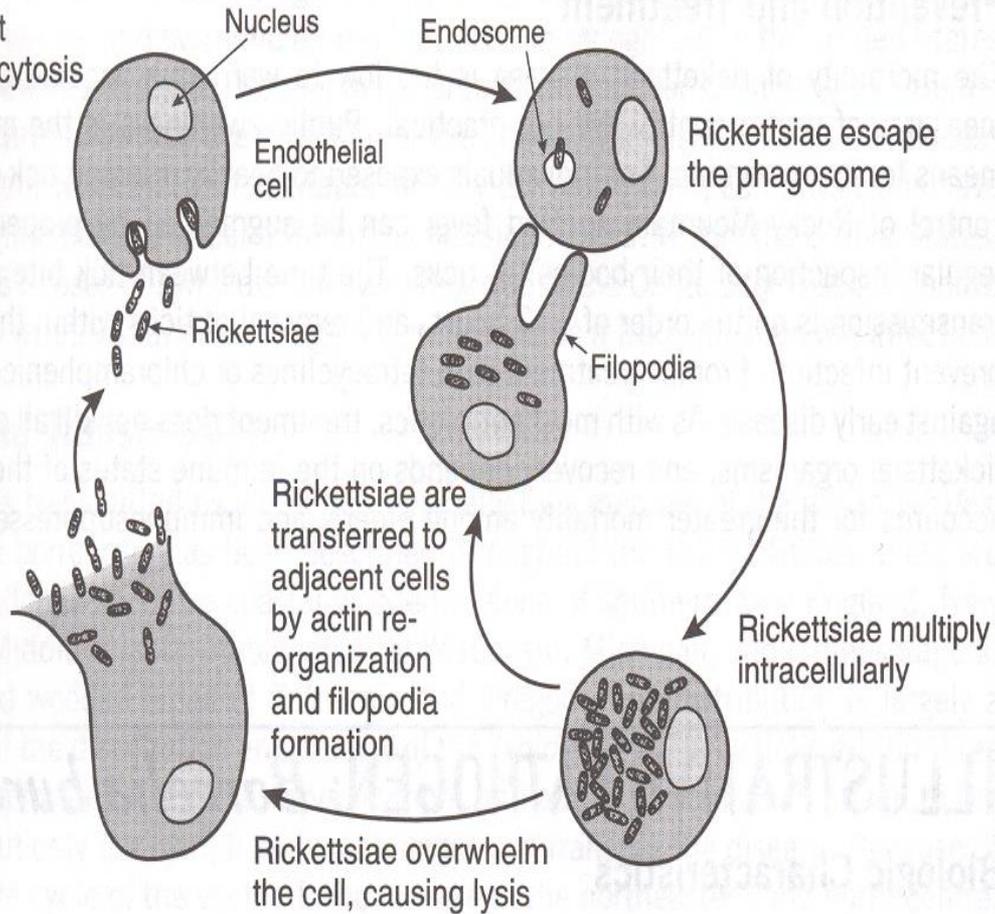


FIGURE 8-3

Cell-to-Cell Spread by Rickettsia rickettsii

Rickettsia (typhus group), clinical picture

- Clinical significance – the diseases caused by *Rickettsia* are all characterized by fever, headache, myalgias, and usually a rash.
 - Typhus fevers – incubation is 5-18 days.
 - Symptoms include a severe headache, chills, fever, and after a fourth day, a maculopapular rash caused by subcutaneous hemorrhaging.
 - The rash begins on the upper trunk and spread to involve the whole body except the face, palms of the hands, and the soles of the feet.
 - The disease lasts about 2 weeks.

Typhus fever

- Two types of typhus may occur:
- Epidemic typhus – caused by *R. prowazekii* and transmitted by human lice as it bites and defecates in the wound.
 - This occurs in crowded areas causing epidemics. Mortality rates are high in untreated cases. Following an initial attack, some individuals may harbor the organism of a latent infection with occasional relapses = Brill-Zinsser disease
- Endemic typhus – caused by *R. typhi* and transmitted to man by rat fleas.
 - The disease occurs sporadically, but is clinically the same, but less severe than epidemic typhus.

Spotted fever group

- Spotted fever group; *R. Rickettsi* transmitted by a mouse mite.
 - After a 1-2 day incubation a papule develops at the entry site of the insect and within 1-2 weeks a fever, malaise, and headache develop followed by a rash.

Rocky mountain spotted fever

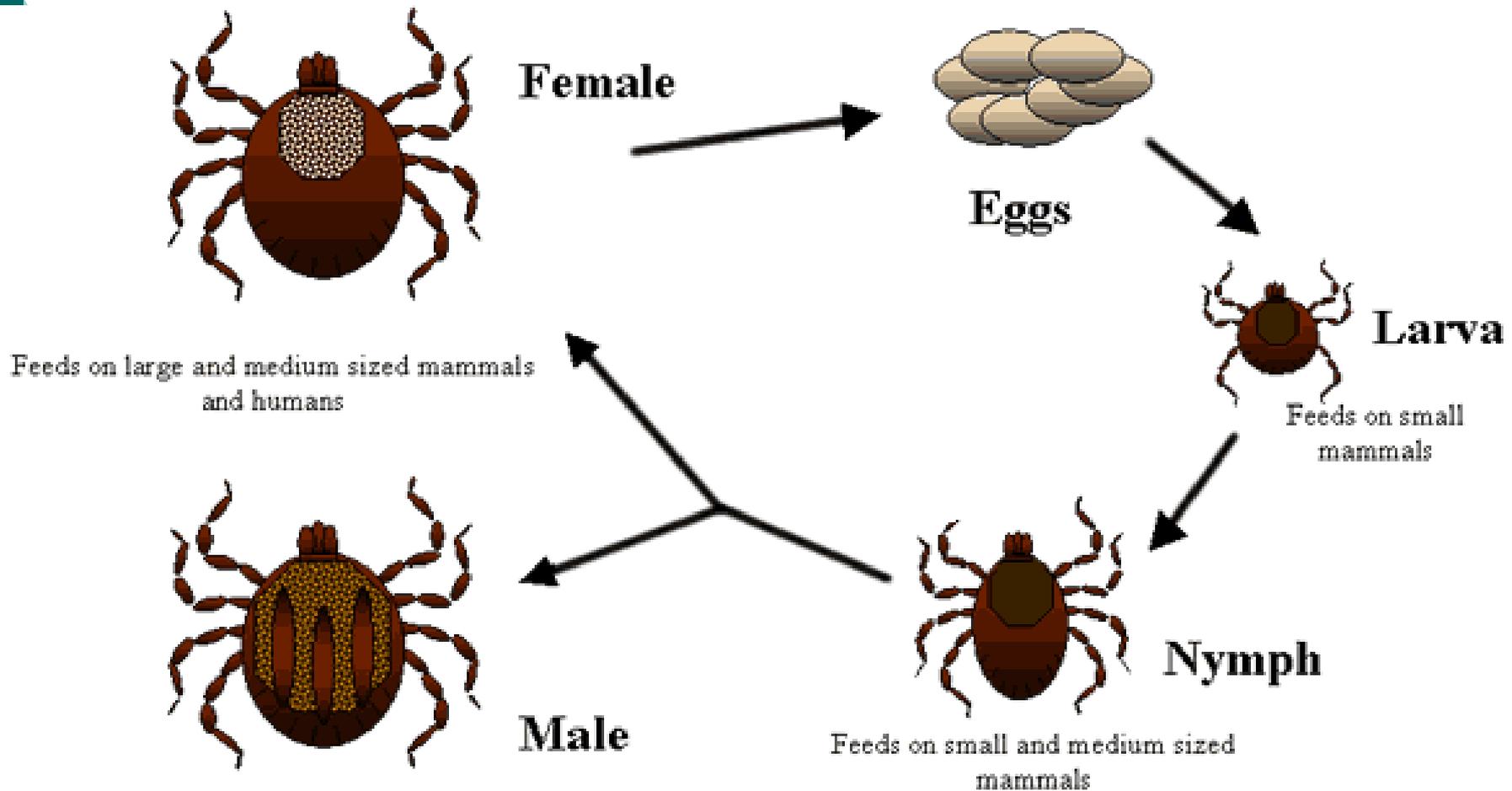


FIGURE 23.17 The rash caused by Rocky Mountain spotted fever. This rash is often mistaken for measles. People with dark skin have a higher mortality rate because the rash is often not recognized early enough for effective treatment.

Rocky mountain spotted fever

- is the most lethal and most frequently reported rickettsial illness in the United States
- The disease is caused by *Rickettsia rickettsii* a species of bacterium that is spread to humans by *Dermacentor* ticks (wood tick) .
 - Initial signs and symptoms of the disease include sudden onset of fever headache ,and muscle pain followed by development of rash.
 - The rash has a centripetal or "inward" pattern of spread, meaning it begins at the extremities and courses towards the trunk . .
- approximately 3% to 5% of individuals die from the infection .

Wood Tick



Complications of *R. rickettsii*

- Because *R. rickettsii* infects the cells lining blood vessels throughout the body, severe manifestations of this disease may involve
 - partial paralysis of the lower extremities ,
 - gangrene requiring amputation of fingers, toes, or arms or legs,
 - hearing loss,
 - loss of bowel or bladder control,
 - CNS; movement disorders and language disorders .
- Host factors associated with severe or fatal Rocky Mountain spotted fever include advanced age, male sex, African-Caribbean race, chronic alcohol abuse, and glucose-6-phosphate dehydrogenase (G6PD) deficiency



Reckettsialpox and Mediterranean spotted fever

- The Other spotted fever diseases associated with red papule at bite site followed by fever muscle pain headache and arthralgia



Scrub fever

- In fewer than half of patients, the site of the mite bite develops a necrotic eschar with enlargement of regional lymph nodes

Diagnosis

- No evident on blood smear and do not stain
- The use of PCR and immunofluorescent antibodies to examine a biopsy is diagnostic.
- The organism can be inoculated into tissue culture but it is unnecessary and hazardous.
- The Weil-Felix test looks for the production of serum antibody that is reactive against *Proteus* OX19, OX2 or OXK antigens but it is not always reliable.

Control

- **Sanitary:** Arthropod and rodent control
- **Immunological:** cox vaccines are killed vaccine. Live attenuated vaccine
- **Antibiotics:** Doxycycline, Tetracycline or chloramphenicol are drugs of choice.



Q-Fever

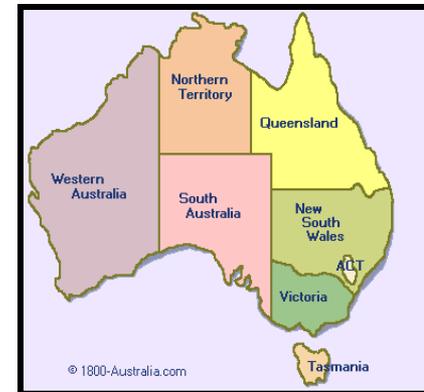
(*Coxiella burnetii*)

Background/History

- **Q fever is a zoonotic disease caused by *Coxiella burnetii*. *Coxiella burnetii* is no longer regarded as *Rickettsiae***
- ***Differ from rickettsiae in that it resist drying, no rash and it is not transmitted by arthropod***
- ***Obligate intracellular, gram negative bacterium***
- **Distributed globally**
- **Found in many species of animals**
- **Highly infectious - one organism may cause disease**
- **Hardy organism**

Background/History

- Q stands for Query or Queensland
- Origin of disease unknown
- First reported cases were in Queensland, Australia



Primary Reservoir

Goats

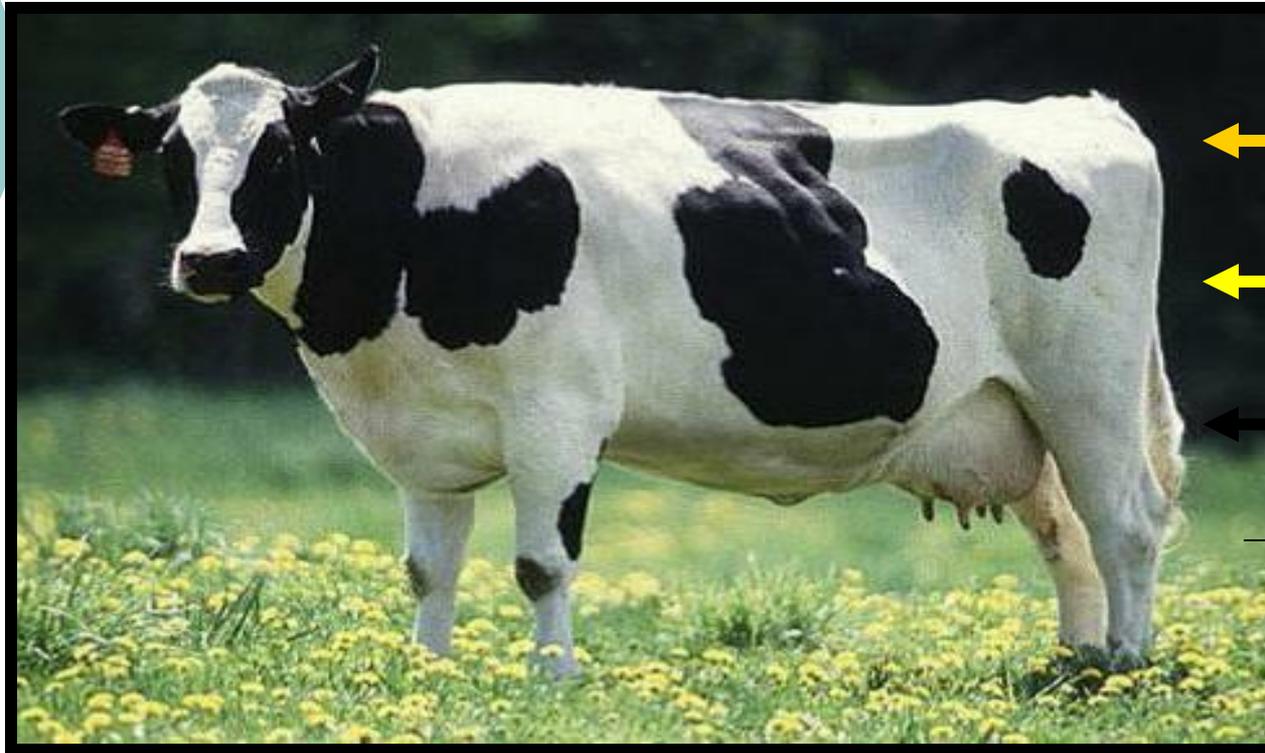
Cattle



Sheep



Bacteria is excreted in:



← Feces

← Urine

← Milk

← **Placenta and
birth fluids**



Survival in environment

- Resistant to heat, drying and disinfectants
- Air samples test positive for 2 weeks
- Soil samples test positive for 150 days
- Spore formation in the air

Transmission

- Most common route is inhalation of aerosols or ingestion of contaminated milk or food.
- Contact with milk, urine, feces and vaginal products of infected animals
- Little from man to man
- Incubation from 9-40 days

Transmission

- Who's at risk?
- Farmers, veterinarians, researchers, abattoir (slaughterhouse) workers etc.
- People who breed animals
- Immunocompromised

Host interaction

Entry via inhalation

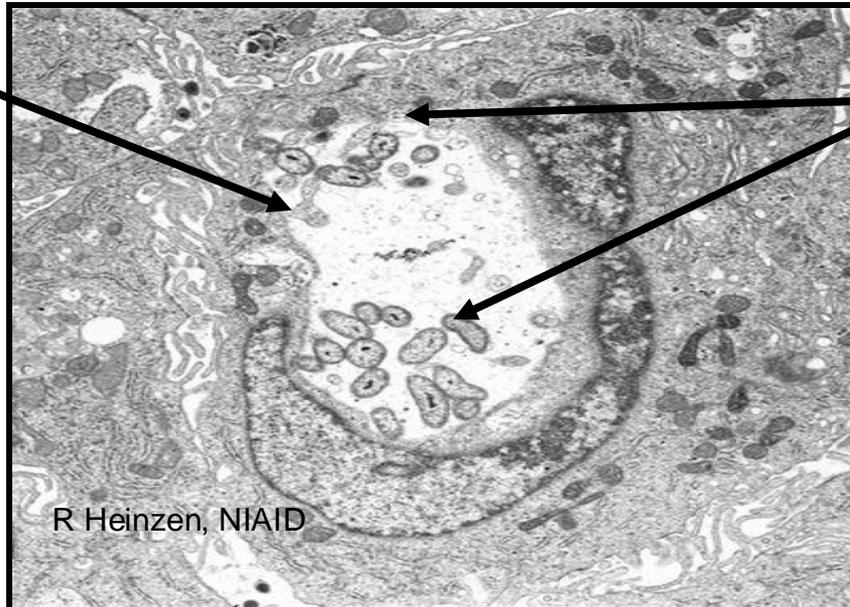
Alveolar macrophages encounter bacteria

C. burnetii phagocytosed and parasitized the spleen and liver

Replication within phagolysosome

Low pH needed for metabolism ,cellular lyses occurs

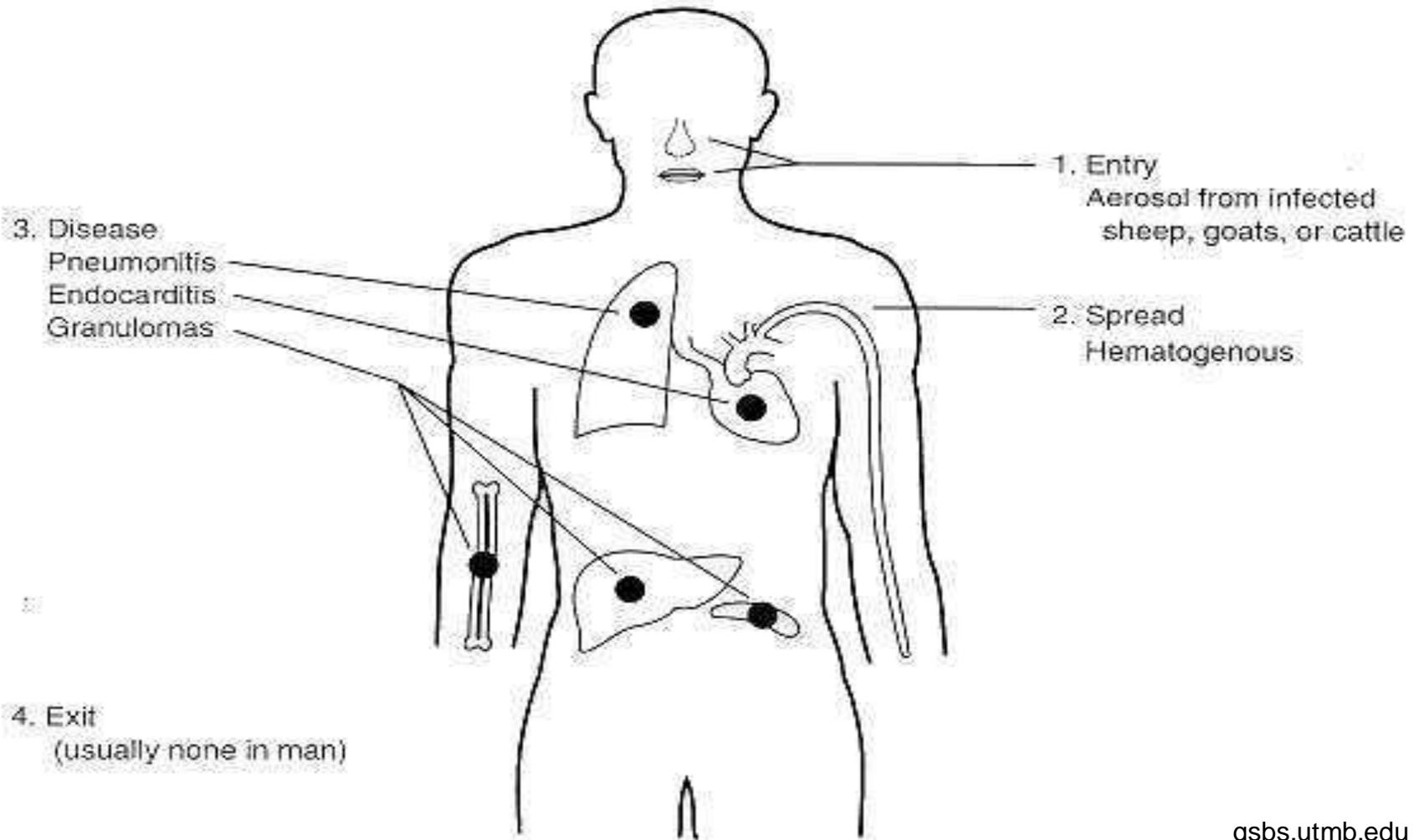
Macrophage



C. burnetii

R Heinen, NIAID

Acute or Chronic Q fever



*Bacteria spread through blood

Symptoms

- Acute Q fever
- Half of the infected individuals exhibit no symptoms, Self-limiting, flu-like disease
- Fever, nausea, headaches, vomiting, chest/abdominal pain
- Interstitial pneumonitis
- Granulomatous hepatitis
- Chronic Q fever (> 6 months)
- At risk are pregnant women, immunocompromized and pre-existing heart valve disease
- Endocarditis & meningoencephalitis
 - Endocarditis results from less than 1% of Q fever cases
 - Endocarditis may develop in 1 to several years after infection

Diagnosis

Hard to diagnose because:

- Asymptomatic in most cases
- Looks like other disease (Flu or cold)
- Serology continues to be best method because it is difficult to be studied outside host (do not reproduce outside)
- PCR, ELISA and other methods
- Liver biopsy to see fibrin ring granuloma
- Weil-Felix test is negative

Diagnosis

- Bio safety level 3 (BSL-3) facility
- Very infectious (one organism causes infection)
- Listed by the CDC as a potential bioterrorism agent.
- Can be Isolated in cell cultures or embryonated eggs





Control/Prevention

- Pasteurization and sterilization with higher temperatures
- Disinfect machines used in farm areas for birthing
- Regular testing of animals and those who work closely with them
- Protective Personal Equipment

Treatment/Vaccines

- Once infected, humans can have life-long immunity
- Acute Q fever treated with:
 - doxycycline, chloramphenicol, erythromycin or fluoroquinolones
- Chronic Q fever treated with:
 - More than one antibiotic
 - tetracycline and cotrimoxazole for 2 years
- Protection is offered by Q-Vax, a whole-cell, inactivated vaccine. The intradermal vaccination is composed of killed *Coxiella burnetii* organisms, protection for years rarely need another dose