Bacteria	Characteristics	Disease	Disease management	Notes
Brucellae	**Morphology** -G –ve coccobacilli -unencapsulated -nonmotile - obligate parasites of animals & humen **Biochemical** -intracellular -aerobic except abortus (requires 5-10% CO2) -complex nutritional requirements (fastidious) -catalase & oxidase +ve -resistant to freezing & drying … but killed by boiling & pasteurization **Culture** -appear predominantly as short coccobacilli in young cultures. -smooth & transparent colony	**Brucellosis (undulant, malta, Mediterranean, Cyprus fever)** -an acute bacteremic phase followed by a chronic stage that may extend over many years & involve many tissues. **Transmission** -unpasteurized milk or milk products (goat cheese) -occupational contact (farmers, vets…) → transmitted by ingestion / inhalation (biological weapon M&S) / skin & mucosal exposure (healthcare providers vs. needle sticks) **Pathogenesis** -enters lymphatics → blood → distribution to different organs → forming granulomatous granules in reticuloendothelial system (brucella here is intracellular) → granules develop into abscesses. -granuloma: contain epithelioid & giant cells , with central necrosis & peripheral fibrosis. -spread in the blood can lead to osteomyelitis, meningitis, cholecystitis. -main histologic reaction is proliferation of PMNs, fibrosis & coagulation necrosis. **Symptoms** -incubation : 1-4 weeks -insidious onset : malaise, fever, weakness, aches, sweats -undulant fever : rises in the afternoon & falls at night متموجة -sustemic effects: -gastrointestinal& nervous symptoms -LN enlargement & palpable spleen -hepatitis & jaundice -osteomyelitis (abnormal standing & deep pain in sacroiliac joint (young age) or low back pain (older) -general symptoms subside after weeks or months ,but local lesions may continue. -a chronic stage may develop after acute infection characterized by aches, low grade fever & psychoneurotic symptoms. (miserable disease)	**Diagnosis** 1-specimens : -blood → for culture -biopsy (LN, bone…) → for culture -serum → serologic tests 2-culture : -brucella agar → specifically designed for brucella Highly inriched (since its fastidious) -trypticase soy medium -chocolate agar …. 3-serology -IgM → rises during the 1st week of acute illness + peaks at 3 months -IgG & IgA → rise after 3 weeks of onset + peaks at 6-8 weeks + remains high during chronic course A-agglutination test → IgG agglutinin titers above 1:80 in active infection . → cholera vaccine may develop false +ve B-ELISA assays → use cytoplasmic proteins as Antigens More specific & sensitive **→ culture needs a long time. So, serology is preferred** **Treatment** -G-ve antibiotics -not easily eradicated due to intracellular location -for best results: treatment must be prolonged. Combined treatment with a tetracycline (eg, doxycycline) and either streptomycin for 2–3 weeks or rifampin for 6 weeks is recommended. **Prevention** -animal vaccine: -B.abortus → live att. S19 & RB51 -B.melitensis → Rev1 -humen vaccine → still experimental -pasteurization of milk	-types : -melitensis → infects goats -suis → swines -abortus* → cattle -canis → dogs → all can infect humen by zoonotic infection (by accidental contact with feces, urine…) -B.abortus is named so bc it can cause abortion to cattles. However , it cant do so for humen (no erythrol in placenta)

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Leptospira	**Morphology (dark field microscope)** -G-ve spirochete (thin, tightly coiled with double membrane) -unencapsulated -motile (2 periplasmic flagella) -question mark appearance **Biochemical** -derive energy from oxidation of long chain F.A & cannot use A.A or carbs. -derive nitrogen from ammonium salts -can survive for weeks in water , particularly alkaline (urine is a great environment) -aerobic	**Leptospirosis** broad spectrum of clinical manifestations → varying from asymptomatic infection (90%) to fulminant, fatal disease (Weil's Syndrome) (10%) **transmission** -main source of infection is animal urine (kidney involvement in many animals is chronic & results in shedding of large numbers of leptospira) -humen urine may also contain leptospira in 2nd or 3rd weeks → transmitted through cuts, abraded skin, mucosa especially oral & conjunctiva. **Pathogenesis** -leptospiremic phase → invasion & hematogenous spread -immune phase → leptospira establish in the liver & kidneys mainly … producing hemorrhage & necrosis (jaundice & nitrogen retention) **Symptoms** -incubation → 1-2 weeks -second phase → starts when IgM Ab titter & manifests as: -aseptic meningitis -nephritis -hepatitis -skin, muscle, eye lesions -LN enlargement	**Diagnosis** 1-specimens: -blood -CSF -urine 2-microscopic examination -dark field -giemsa technique 3-culture -aerobic conditions at 28–30 C in semisolid medium (eg, Ellinghausen-McCullough-Johnson- Harris EMJH) in 10 mL test tubes with 0.1% agar and 5-fluorouracil. -growth is slow (you cant depend on it) 4-serology -microscopic agglutination test (MAT) -ELISA **Treatment** -mild leptospirosis → oral doxy, ampicillin or amoxicillin -severe (wiles, hepatitis, nephritis) → IV penicillin as soon as possible (leptospira has penicillin binding proteins) Immunity → serovar specific immunity after infection (but infection with other serovars is possible). **Prevention** -control of rats (main reservoir) -avoid exposure to urine (especially sewer workers) -avoid exposure to tissues of infected animals -vaccine → for animals	-types : -pathogenic → interrogans -free living → biflexa **Epidemiology** Distributed worldwide … most commonly in tropics & subtropics (climate & poor hygiene)

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Mycobacterium TB	**Morphology (ZN stain – Koch stain)** -red snappers (bacilli) -unencapsulated **Biochemical** -slow growing -obligate aerobe -facultative intracellular -acid fast -non spore forming -non motile	**Tuberculosis (consumption disease/ white plaque- pallor)** -2 conditions: -latent TB (LTBI) → no symptoms , not infectious -TB disease **Transmission** -airborne disease primarily -unpasteurized milk -direct inoculation … → TB enters the lung → dissemination into other parts of the body OR : contagious spread from adjacent lymphadenopathy Primary involvement of extra-pulmonary organ (ex. abdomen) **→ abdominal TB (5% of cases)** -not as common as pulmonary TB -significant mortality due to late diagnosis (nonspecific symptoms) -4 forms : -TB lymphadenopathy -peritoneal TB -GI TB -visceral TB (involving solid organs) **Pathogenesis of GI TB** -occurs via: -reactivation of LTBI -ingestion of MTB (ex. Unpasteurized milk, undercooked meat) -hematogeneous or contagious spread during active pulmonary or miliary TB (ex. retrograde spread from fallopian tube, lymphatic spread) → infection of the mucosal layer of the GI → formation of epithelioid tubercles in the lymphoid tissue of the submucosa → (after 2-4 weeks) caseous necrosis & ulceration (painless) of the mucosa → infection spreads into deeper layers → spread into adjacent LN → spread to the peritoneum →rarely, bacilli enters into the portal circulation & involve solid organs like liver, pancreas & spleen. **Symptoms of GI TB** -abdominal pain & general compliants (non specific) -obstruction, hematochezia, palpable mass … -fever, weight loss , anorexia , night sweats. -any part of the GIT can be infected … most commonly, terminal ilium & cecum **→ generally symptoms depend on whether the infection is pulmonary or extra pulmonary … fever & night sweats are common symptoms**	**Diagnosis** 1-smear microscopy -3 specimens should be taken from each suspected Patient → examined microscopically by -ZN stain -yellow fluorescence after auramin stain 2-culture -Both liquid and solid mycobacterial cultures should be performed for every specimen. - recovered isolates should be classified according to standard criteria → - (Lowenstein-Jensen or Middlebrook 7H10), - Radiometric broth culture (BACTEC radiometric system) - Mycobacterial growth indicator tube (MGIT). -culture is the GOLD STANDARD + it allows doing susceptibility tests for the causative agent. -however , it takes a very long time 3-nucliec acid amplification test (NAAT) 4-tuberculin skin test (TST) → common false +ve 5-interferon gamma release assays (IGRAs) → positive results in 4& 5 can be due to previous exposure (false positive) **Treatment** -Active TB → drug cocktail (4 drugs) -isoniazid -rifampin -pyrazinamide -ethambutol or streptomycin -Latent TB → -isoniazid preventive therapy (IPT) -مشكلة العلاج انو مدته طويلة و المرضى ما بلتزموا خصوصا انو الأعراض بتروح بعد اسبوعين **Prevention** -diagnose & isolate infectious cases rapidly -administer appropriate treatment for the appropriate period. -treatment of pts with LTBI at high risk of activation -vaccine : BCG vaccine → -attenuated vaccine derived from M.bovis -low efficacy rate (0-80%)	-MTB can cause disease in humen & other livings -types : M. tuberculosis (Mtb) Mycobacterium africanum Mycobacterium bovis Mycobacterium microti Mycobacterium caprae Mycobacterium pinnipedii Mycobacterium suricatte Mycobacterium mungi Mycobacterium dassie Mycobacterium oryx Mycobacterium canetti **Epidemiology** -1/3 of the population have latent TB … only small proportion become sick -leading cause of morbidity & mortality