Resume :

Bacteria airborne: Most of respiratory bact are gram + Strptocci: aerotolerant homofrementative for lactic acid, gram + , no spores, catalaze -, phylum firmicutes, form chains, a- Pyogenes (**β-hemolytic**): red blood cell hemolysis . b-Viridans subgroup (**α-hemolytic**): many Streptococci, Lactococci and Enterococci do not produce hemolysins but instead cause the formation of greenish or brownish zones around colonies on agar (hb to methemoglobin)

**Strp pyogene**: pharingitis skin mamalary ear infection and scarlet fever because of superantigen exotoxin A from lysogenic phage, can cause rheumatoid bacuse m protein similar to heart and glomuronephritis. Virulence factors: homolysin s(non imugenic) and o (immugenic ),streptokinase, c5a peptidase, mprotein (binds factorh which degrade c3b) and exotoxin A. Cpsule help evede immune system.vaccine coming soon

**strp pneumonia**: lung infection, and other infection, virulence by capsule which protect it from immunity, 91 serotype of capsular polysaccharides(linked to pgn with phosphate diester bonds), can got to blood and other organs. Vaccine for 2/3 of polysaccharides. Given for people succsseptible.

**Corynebacterium diphteriae:** It is a Gram +ve, non-motile, rod-shaped, aerobic bacterium that forms irregular shaped, club-shaped or V-shaped cell arrangements, belongd to actinobacterium (filaments to rods), common inhabitants of soil and plant materials, skin diphtheria (forms papula then ulcer from skin wounds), respiratory dipht (pseudomembrane composed of bacteria, lymphocytes, fibrin, and dead tissues, covers the tonsils). Toxin is its ab extoxin (adp ribosylate elongation factor 2 carried by tox gen in phage beta). Has a vaccine from the neutralised toxin part od dtp.

**Mycobacterium tuberculosis:** +ve, non-motile, rod-shaped, aerobic bacterium part of actinobacter,myco=fungus like because filamentous, rope wrinkled like arrangement because of glycolipid cord. Acid fast.Slow growth g=20h .Resistant to drying and disininfectant and antiseptic bcs of the mycolic acid. Mostly in immunovompetemt ppl pneumonitis, tissue distruction causing bloody sputum, phocytose by alveolar macrophage which inactivate by inhibiting phagosome lysosoime fusion. Vaccine from myco bovi (bcg strain)

**M. leprae:**many ppl are resistant not grow on art media,infect macrophage and schwan cells, lepromatous from( lesion on skin), tubrculoid(less severe, strong lymp t reaction)

**Bordetella pertussis:** Gram –ve, obligate aerobic coccobacillus belonging to the class Proteobacteria (all are Gram - with great metabolic diversity), Bacteria initially grow on cilia and lead to their destruction and whooping cough (accumulation of mucus) after symptoms of cold. Incubation period is 7 to 10 days. Virulence: protein adhesions (pertactin, hemaglutinin), petrussiin toxin(a5btoxin, adp ribosylate Giα of adenyly cylase leading to camp production increasing respiratory secretion and mucus), adenylate cyclise (adenylate cyclase/hemolysin ) and tracheal cytotoxin (produced by all gram - , toxic to ciliated cells). Vaccine in dtp from whole inactivated cell.

**Neisseria meningitides:** aerobic, Gram –ve coccus typically arranged in pairs, catalase and oxidase positive.13 subgroupss based on capsular plysacharids (only 5 pathognic). attaches to the cells in the nasopharynx, causes meningitis (infection of meninges of the cns membranes) carriage is typically transient and the bacteria is cleared after specific antibodies develop)(epidemic spread of the disease results form the introduction of a new virulent strain into an immunologically naive population) virulence: pili, los (lps with no o antigen involved in invasion of endothelial and epithelial), opa (integral outer-membrane invasion protein, opa hs and opa ceacam, mutation, expression of each Opa protein can be independently switched on and off), polysaccharide capsule (invasion, anti bactericidal and anti phagocytic, porins(por b ca influx and apopotosis). Vaccine (not long term) from capsule in epidemics, antibiotics as prophylaxis available.

**Vaccine MMR:** paramyxovirus: measles (viremia +rash+fever+infections) and mumps( infection of salivary gland with swelling in jaws and neck,go by blood to brain testes pancreas, quick recovery with antibody. Rubella togavirus like attenuated measles, dangerous to pregnant women.

**Varicella:** chicken pox by varicella zoster virus, systemic papular rash, that quickly heals an attenuated virus vaccine is available. Can remain dormant in nerve cells then causes shingles in immunocompetent ppl. Hyperimmune globulin prepared against the virus are used to prevent symptoms of shingles.

**Influenza A:** 16 HA and 9N subtypes in humans are H1N1, H1N2, and H3N2, infects the mucous membrane of the respiratory tract and occasionally infects the lungs . avian ha binds 2.3 in respiratory and intestinal epithelium. Human infect 2.6 in upper respiratory tract. (2.3 on ciliated and 2.6 on none ciliated in humans). The HA protein is synthesized as a precursor protein that is cleaved into 2 subunits (HA1 and HA2) by host cell proteases. This cleavage is a prerequisite for fusion of the viral and endosomal membranes and, therefore, for viral infectivityi- Low pathogenic avian influenza viruses possess a single Arg residue at the cleavage site, recognized by extracellular, trypsinlike proteases (found in respiratory and intestinal tract)ii- By contrast, highly pathogenic avian viruses (e.g. H5N1) possess multiple basic amino acids at the cleavage that are recognized by ubiquitous, intracellular, subtilisin-like proteases that, thus, trigger systemic infection. We also need a drop in ph. most of the serious consequences of influenza infection occur from bacterial secondary infections in individuals whose immunity has been lowered by influenza. Tamiflu inhibits neuraminidase, vaccine present but not reliable bcs a lot of strains.

**Direct contact diseases**

**Staphylococcus:** facultative anaerobic, Gram +ve cocci, catalaze +ve, that form grape-like clumps,dry resistant,infect skin and wounds, from normal flora of assymptomatic to immunocompetent. There are s.epidrmidis and s.aureus. causes various infection pneumonia arthritis. Virulence: capsule 11 serotypes and slime layer (protects from phagocytes and allow adherence),protein A (affinity for fc region on igg inhibit phagocytes and induce formation of compexes outside cells), hyaluronidase (hydrolyses matrix and produced by majority), lipases (invades cutaneous and subcutaneous), cytolytic toxins (α, β, δ,γ and Panton-valentine that are cytolytic), enterotoxins (act as intestinal enzymes and thermal resistant superantigen, increase inflammation permeability of intestine with diahrea and vomiting, made by 30-50% ), toxic shock syndrome toxic 1(proteolysis and heat resistant, act as superantigen and induce diarehea and fever). No vaccines a lot of people are carriers.

**Helicobacter pylori:** a gram-negative, highly motile, spiral-shaped bacterium. It has 1-6 polar flagella at one end, infect stomach and causes gastroenteritis ulcer and lead to carcinoma, its protected in the mucosa of the stomach. Virulence : urease and urea transporter (at low ph urei allow urea to go in and cleaved by urease to bicarbonate and ammonia to neutralise medium), vac A (important for helicon for invasion, Vac A causes vacuolation of eukaryotic cells; damages mitochondria (hence reducing ATP synthesis and triggering apoptosis); activates intracellular signalling pathways, increases transepithelial flux of certain molecules, including urea (most probably through Vac A channels); alter the activity of immune cells the secreted toxin can assemble into water-soluble oligomeric structures, and can insert into lipid bilayers to form anion-selective membrane channels, secreted through t5ss) cag A(secreted through t4ss, disrupts epithelial junction and induces morphological changes in epithelial cells), adhesins.

**Hepatitis:** a-e and g differ in replication, transmission, mode of the disease and time course and structure, all infect hepatocytes. Hav is detergen, acid, temperature, salt resistant, non fatal causes mild symptoms, comes from fecal contaminated oysters. It only cause immune inflammation in the liver. Symptoms: fever, nausea fatigue and jaundice, milder symptoms in children, shed in feces after 14 days. Long live immunity is given. Vaccine of killed hav is present , chlorinated water kill it.

**Sexually transmitted diease:**

**Nisseria gonorrhoea:** Gram - obligate aerobe, catalaze positive, oxidase positive diplococcus, fastidious like meningoccocus, sensitive to dryness and sunlight, need to associate wit urogenital membrane. Female:mild symptoms and males have pain in urethra. But can lead to joint, genital and heart damage because of immune complex. Cna cause eye infection in newborns. Virulence: Fimbriae/type-IV pili, porin:form channels for nutrient for bacteria porb : interfere with the phagosome-lysosome fusion in neutrophils, iga1 protease(Produced by N. meningitidis and N. gonorrhoeae. It cleaves the hinge region in IgA1, inactivating the Ig.), opa proteins ( bacterial adhesion, multiple gene for one isolate, ligands for opa are hs receptor like syndican and ecm proteins., cell surface receptors (binds transferring and lactoferrin. No vaccine, safe sex mesureneeded , long term immunity because of antigenic variation of Opa and pili.

**T. pallidum**: is a gram-negative, micro-aerophile spirochete long slender, it has 3 axial filaments (endoflagella) inserted at each end, which is typical of spirochetes, only human reservoir cause of syphilis. Has no lps, rare outer proteins, a lot of missing metablic pathways, can come from oral and skin abrasion but mostly from sex, primary syphilis: lesions where it entered, that disappear giving relief . segondary: disseminate in blood giving rash and flu symptoms. Late syphilis: many tissue infected after years. Can penetrate placenta and cause disease.

**Chlamydia trachomatis**: obligate intracellular Gram-negative, coccoid bacteria, lack pgn but om, differences in om proteins generate different sereotypes. cycle (48hrs).Elementary body:0.3 microns, small round, pear shaped, electron dense, no pgn, protein with disulfide bridges, infectious stage that tolerate outer cell. Reticulate body: intracellular replication, no pgn, metabolically active have all the pgn enzymes though. target cells include non-ciliated epithelial cells found on the mucous membrane of the urethra, fallopian tubes, respiratory tract and conjunctivae, can also infect phagocytes, infect cell and destroy them. Can cause trachoma( scarring and vascularisation of cornea repeated infections with different serotypes of C. trachomatis.cause blindness especially infants), infection of genitourinary system, infant pneumonia. Trachoma is a disease of the poor world. it is the second cause of blindness after cataract. No vaccine.

**Animal and arthropod borne diseases**

**Rhabie:** belongs to Lyssavirus, -ssrna virus , family rhabdovaridae,leads to encephalitis after inflecting cns neurons(by the attachment of the G protein to several receptors including the nicotinic acetylcholine receptors), reservoirs for rabies include raccoons, coyotes, foxes, dogs and bats. It starts from the bited from the saliva of the animal replicate in muscle and connective tissue then ascend to cns(where can’t be reached by immune system) by peripheral nerves.symptoms: fver excitation, dilation, hydrophobia.death occur from respiratory paralysis. Domestic and wild wild vaccination from g protein

**Hantavirus:**3 sements**,** replicate like influenzabunyvaridae(most are arbovirus exept for hanta) related to ebola and lassa (all cause hemmorhagic fever with organ failure).Reservoir: mice,rats, lemmings, vole and others. Transmitted from inhalation of feces or urine or bites. Replicate in pulmonary endothelial cells and small capillaries. Cellular receptor are β3 integrins expressed on platelets, endothelial cells, smooth muscle cells, Macrophages and DCs.

**Rickettsias**: are small, gram-negative (cell wall have 3 layers +S layer) cocci or rod-shaped bacteria they are obligate intracellular (that’s why need arthropods) parasites . absence of host cells. Able to transport Atp, no glycolisis genes, but have complete respiratory genes and can use nadh e transporter,have pyruvate dehydrogenase and puruvate transporterfor tcA,no amino acids or riboflavin, vitamin B6 and nicotinamide or nmp synthesis. This is erosion. Infection from bite (feces or saliva). Infect endothelial cells and can cause blockage and destruction of vessels. **Rickettsia prowazekii:** can use tca, causes typhys, human are reservoir and transmitted by Pediculus humanus corporis which dies from it after 2-3 weeks.they are phagocytose by endothelial cells, have phospholipase taht degrade phagosome, the replicate and lyse cell, infecting other cells, causing edema and lost of vessels, causes fever and rash expt in hands and soles. May lead to all organs failure. Found in Cool region of the south. Vaccine of inactivated bact is found. **Rickettsia rickettsii:** causes mountain spoted fever,transmiteed by wood and dog tick which must stay for 24 hours. After being waked by the warm blood, and coming from salivaCells of R rickettsii grow within the nucleus as well as in the cytoplasm of endothelial cells and are continuously released from the cells by an actin-based motility, do like prowazekii but rach include palms and soles. Mortality 20%. Reservoir are rodents.spread in south and central America. Go from 1 genration to others through eggs. It’s lethal for the tick itselve.

**Borrelia Burgdorferi*: deer*** tick (***Ixodes scapularis )***borne,gram – spirochete , microaerophile, have 7 -20 endofalgella on each pole for twisting motility, 10 sp. Cause lyme disease. Reservoiras are feild mice, infect various animal and occasionally humans, causes a rash and red spot then turn to flu symptoms, can lead to different complication (heart disease arthritis). Vaccine are available.

**Malariae :** 1 millions death/ year from children in subsahara. In poor countries of the south, from anopheles gambia, human is reservoir,. Micronemes: organelle secretes proteases and adhesion for motility and invasion. Rhoptries: club shaped organelle that help in invasion and main contributor in parasitophorous vacuole. Dense granule:post invasion organelles responsible for pv in indected cells. Exoneme: involved in egress befor microneme, have proteases. P. Falciparum virulence: cytoadherence of infected RBC to endothelial cells of the blood capillaries, and to non-infected erythrocytes forming rosettes. This induces inflammation and block the tissue capillaries causing low oxygen transfer to tissues cytoadherence is mediated by pfemp1 which can bind to chondrotin sulfateA(present in endothelial of a pregnant women, can cause anemia and alter nutrient exchange, cd36… It ‘s encoded by 60 var genes. Malaria only express 1 pfemp, and changing from pfemp1 to another let it evade immunity. Symptoms : flu like and fever 40 degress, diarhea and vomiting, and enlargement of the spleen and anemia. Oluging of vessels in brain cause cerebral malaria, can cause kidney failure because of released haemoglobin. No vaccines,drug really expensive,

**West nile Virus:** enveloped virus, with a + ssRNA genome, flavivirus group, transmitted mainly by culex and other mosquitoes, more than 200 birds are reservoirs, humans and horse are dead incidental ends. Mostly assymptomatc to mild symptoms, can in 1 % cause encephalitis. No vaccine for human yet but present for geeses and horses.

**Dengue virus:** flalivirus too, cause by aedes aegypti and albopictus, endemic in mor than 100 tropical country, 4 sereotypes den 1-4, infection with one doesn’t confer immunity to the other, human is the reservoir, replicate in lymph nodes then through blood to organs, causes pain and fever and rash. Re-challenge with other strain cause hemorrhagic fever (decreased platelets and increased vascular permeability)

***Yersinia pestis:*** causes plague, Gram-negative, facultative aerobic rod , human get it by accident, rat reservoir transmitted by rats flea *Xenopsylla cheopsis*, an infected flea is always hungry because of regirgigation of the food, Bubonic plague:75% death after 7 days, fever than buboes (inflammatory swelling of lymph nodes in groin or axilla),then bacteremia, Multiple hemorrhages produce dark splotches. Pneumonic: causes bloody suputum , 90% death. Virulence: adhesins(attach to macrophages and ecm (llaminins),some have protease ativity cleave complement and inhibit coagulation),Yop virulon: extrachromosomal 70 kb plasmid encoding the Yop effector proteins, as well as a type III secretion system ()yop h(, yop T, yopp/j, yop E.control of infected animalsis needed. **YopH:** (protein-tyrosine phosphatase) It dephosphorylates Pi-tyrosine containing proteins in macrophages, hence compromising their immune functions such as:a- inhibition of actin-cytoskeleton rearrangements hence blocking phagocytosis b- downregulation of the inflammatory response by inhibiting the production of chemokines, hence inhibits the recruitment of other macrophages to the site of infection. **YopT:** (a cysteine protease) It cleaves RhoA, Rac1 and Cdc42 (Rho family of GTPases) disrupting actin cytsokeleton rearrangement and hence phagocytosis **YopP/J:** (a cysteine protease) Inhibits intracellular signalling pathways in macrophages, hence blocking production of inflammatory cytokines and chemokines (recruitment and activation of macrophages and other cytotoxic cells to the site of inflammation is abolished)**YopE:** Also interferes with the actin cytoskeleton.

**Waterborne:** the most important pathogenic bacteria transmitted by the water route are *Vibrio cholerea* and *Salmonella typhi* Ten vibrio are implicated in human disease Vibrio cholerea, Vibrio parahaemolyticus and Vibrio vulnificus most are halophile except for cholera

**Vibrio Cholera:** a Gram-negative, curved (comma-shaped) rod,poor countries,causes diarehea, can grow in a broad temperature(14-40), tolerate acid but not gastric acide hence we need a lot of it (1to the8) to initiate infection. If gastric acid is neutralise 10\*4 will do. Most vibrios have a polar flagellum and are oxidase positive and all have lps. 140 serogroups (O1-O140 o-polysacharides)all exept o 140 causes epidemics and secrete choleara toxin. Virulence: Colonization factor (adherence), toxin co-regulated pilus(tcp gene,adherence to intestinal mucosa, receptor for the cholera toxin carrying lysogenic bacteriophage CTXΦ), zonnula accludens toxin(increase intetin permeability by loosening tight junction), accessory cholera enterotoxin(increase fluid secretion) , cholera toxin(A5B, B subunit binds to the GM1 ganglioside, A adp ribosylate Gα that activate adenylyl cyclase that converts ATP to cAMP , induce the secretion of potassium, chlorine and bicarbonate ions from the cells into the intestine lumen. 20 l of fluids can be lost.Mortality 50%. Comes from water, shelfish and vegetable contaminated by sewage. Watery diahreha and vomiting and rice water diarhea. Variety of vaccines but no long term protection. Avoid raw food and well treatment of sewage.

**Legionella pneumophila***:* a thin, gram-negative, obligate aerobic rod.48 species of legionella but pneumophilla is the major infective.need iron and l- cysteine. the pathogen grows in water and is disseminated in humidified aerosols. Human infection is via airborne droplets but infection is not spread person to person. Thermotolerant. Present in lakes and ac. It’s intarcellular , macrophage and monocytes and in nature amoeba. Phgocytosed by binding c3b to porin but remain alive by inhibits the phagolysosome fusion and proliferate in the intracellular vacuole producing proteolytic enzymes, phosphatase, lipase and nuclease which kill macrophage that can be activated by sentisised t cells. Can be assymptopatic. Pontiac fecver: resolves on it on , fever and malais. Legoinaire : more severe, pneumonia and multiorgan disease. Heating and chlorinating water is enough to kill it.

**Giardia** (destroys the microvilli of intestinal cells causing a foul-smelling diarrhea), **cryptosporidium(**grows intracellularly in mucosal epithelial cells of the stomach and intestinecausing diarhea), cysts have a thick protective wall that allows the parasite to resist drying and uv and chemical disinfection or chlorination we need to have filter and heating water.

 **amoeba(***Entamoeba histolytica*  is an important human pathogen which grow both on and in the intestinal mucosal cells and cause extensive tissue damage leading to diarrhea and abdominal pain). Mostly tropical and subtropical use chlorination and filters. Viruses like polio and hep A are kille by clorination.

**Foodborne**

**S. Aureus:** 7 differnt enterotoxin, most produces is A,stable at 100` , resistant to hydrolysis by gastric and jejunal enzymes, causes gastroenteritis. Store at low temperature to inhibit bacterial growth+hygiene.

***Clostridium perfringes*:** anaerobic, gram-positive rod, spore forming, haemolytic, grows rapidly, found in soil, large doses needed in all kind of meat, perish in gatric acidity, the one that stays infect ppl and sporulate.Classified A-e according to toxin productio .virulence: Alpha toxin( produced by all, lechitinase phospholipase c attacks phosphatidyl choline on all blood cell and endothelium, causes homolysis,vleed, vascular permeability and tissue destruction.Beta- tocxin(necrosis, lysis of intestinal epithilia.)Epsilon toxin(protoxin activated by trypsin, it’s taken by blood vessels and increase permeability by lysis of endothelium and causing edema) Iota toxin (necrosis and increase vascular permeability), enterotoxin ( produced by type A,improved by trypsin, heat labile, superantigen, it produce by sporulating cells, i alter permeability of intestinal epithelium by forming complex on the membrane. Soft tissue: go through wounds, can causes gaz gangrene, food poisoning because of ingestion type A. Necrotising enteritis and perforation in the intestine causisng peritontis(abdominal symtoms because of B-toxin). Can be prevented by eound care and hygiene.

**C botulinum :** botulinum toxin induce pathogenisis, toxins ( A-G), Abef toxic to humans. Ab toxin, A chain has a zinc-endopeptidase activity cleaving protein involved in the exocytosis of acetylcholine at neuromuscular junction causing flaccid paralysis death with respiratory parakysis, escape lumen by tancytosis into epithilia and interaction with auxiliary protein that protect it from enzymes, can cause infant botulisim by colonizing infant guts which isn’t possible in adult because of norma florausually from honey, wound botulinisim from bacteria on wound and foodborne toxin have same symptoms. Heating at 60 to 10min. Children should not eat honey.

**Slamonella:** gram-negative, facultative anaerobic rods (catalase positive and oxidase negative.) can infect all animals, especially faces and sewage. *S. typhi* causes the disease typhoid fever only in human. Infection from eggs dairy poultry infected surfaces. Virulence: fimbriae (bind to microfolds in preyer patchers), T3ss(spi-1 secrete protein that induce rearrangement of actin in m cells and subsequent phagocytosis, spi-2 facilitate replication in slamonella containing vacuoles, tolerance to acids(at least 50 genes in vacuole and stomach). Can gostroentiritis that spontaneously resolve, can cause septicimia because of bacteria in blood especially in weak immunity, enteric fever with gradually increasing fever this is the typhoid fever. Food shoulbe heated at 70 for 10 min.

**E.coli:** esherishia is 5 species, coli the most spread,catalase positive,oxidaze negative flagellated, gram-negative, facultative anaerobic rods,200 strains pathogenic, become so by taking plasmids, pathogenicity island or bacteriophages. Cause diarrheal disese and urinary tract infection. Virulence: 1) Adhesins:use it to attach to urinary and gastric epithelium not to be flushed, for adhesion they also use fimbria and pilin and colonisation factors.2)Exotoxins: shiga toxin(stx1 and 2, both are a5b, b binds to fglycolipid receptor of intestinal ivllus and renal endothelial, a is cleaved to A1 and A2, A1 binds 28s rRna and distrupts protein synthesis, they distrcut villus and increase fluid secretion), heat stable toxin (sta, binds and activate guanylyl cyclase in brush border epithelium increasing cgmp and fluid secretion by changing ions permeability), heat labile toxin (Lt1, a5b toxin, b bind to gm1 ganglioside and other receptor in intestine, A adp ribosylate gialpha and increase camp and alter ions permeability leading to watery diahrea. 3) microvili effacement:first EPEC cells express bundle-forming pili (Bfp), the intimate adhesin intimin, and short, surface-associated filaments EspA filaments, and then th interaction with epithelium and insertion of tir which get phosphorylated and inserted on host surface, tir is secreted by t3ss along with other molecules that affect intracellular signalling leading to depolymerisation of actin and loss of microvili. Tir and intimin bind leadin to accumulation of actin and othe cytoskeleton element beneath the cell which result in formation of pedestal structure which lead to distruption of host processes, lost of tight junctions, electrolytes loss and cell death.Epec can lead to gastroenteritis by diarhea especiallay in poor countries from malabsorption, adulkts have protective immunity. ETEC gastroenteritis common in poor countires ETEC bacteria produce heat-labile (LT) and heat-stable (ST) toxins. Ehec gastroenteritis caused by shiga toxin,they cause a/e lesions on epithelial cells,have plasmid that provide other virulence factors, causes various forms of diarhea, shiga toxin stx2 can destroy glomerular endothelial cells resulting in renal failure. Rare EIEC gastroenteritis also diarrhea , it incodes factors that mediate invasion of colon in vacuoles which are lysed and they replicate in cytoplasm and move to other cytoplasms using actin tails. Urinary tract infections: ecoli originate in colon go to urethra bladder and the kidney or prostate almost all ecoli produce uti.

**Campylobacter:** gram-negative, motile, curved rod-to-spirillar shaped organisms miroaerophiles, there are 2 strain jejuni and coli that causes diarhea after invading the intestinal epithelium. Jejuni is normal resident in poultry and turkey and sensitive to gastric acids that’s why a big dose is neede more than 10 to the 4.

**Listeria monocytogenes:** gram-positive, that is acid tolerant, psychrotolerant (cold-tolerant) and salt-tolerant, facultative aerobe, found everywhere,soil water, refrigeration isn’t effective. Can have assumptopatic or mild flu. Meningitis occurs withweak immunity, neonatal disease high mortality in new born from mothers by placenta. Hence 3 barriers (placenta, intestine and blood brain. In cytosol it forms segondar vacuole to invade neighboring cells and propel itself through actin tail. Internalins: surface protein that mediate invasion via n terminal leucine rich repeats which interact with glycoprotein receptors, internalin A(c terminal attached to pgn and n terminal to E-cadherin glycoproteinon epithelium and placenta) , internalin B(c terminal bound to lipoteichoic acid and n terminal bind to receptors for Inl B include a receptor tyrosine kinase on hepatocytes and gC1q receptor (ubiquitously expressed). **Listeriolysin O (LLO)** (aided by phospholipase c and the ph of phagosome 5.5 to 6.0, it can form a pore in primary and segondary vacyole, llo is a receptor for cholesterol. ActA protein localised on surface of bacteria activate arp 2/3 a seven protein host complex that allow intracellular movement by polymerisation of actin at bacterium actin interface.

Foodborne infections may be caused by other bacteria such as *Yersinia enterocolitica* (gram –ve rod) *and Bacillus cereurs* (Gram +ve, endospore forming)

* The largest number of annual food borne infections are thought to be caused by viruses. viral food-borne illness consists of gastroenteritis characterized by diarrhea, often accompanied by nausea and vomiting. recovery is spontaneous and rapid usually within 24-48 hrs (known as the 24-hour bug)
* Important foodborne protozoans include Giardia lamblia, Cryptosporidium parvum and Toxoplasma gondii. (all these parasites can be spread via food, presumably contaminated by fecal matter in untreated water)