Microbiology summary

	Gram positive	Gram negative
Gram reaction	Stain blue or purple	Stain red or pink
Peptidoglycan layer	Thick (multi-layered)	Thin (single-layered)
Teichoic acids	Present in many	Absent
Periplasmic space	Absent	Present
Outer membrane	Absent	Present
Resistance to physical disruption	High	Low
Resistance to drying	High	Low
Antibiotic resistance	More susceptible to antibiotics	More resistant to antibiotics
Conjugation systems	Present	Present
Transformation of plasmid	More	Less
Transformation of bacterical chromosome	Yes	No

Microbiology summary

Drugs that inhibit cell wall synthesis: Beta lactam drugs (Bactericidal)		
Penicillin	**Penicillinase resistant drugs**	**Cephalosporins**
Narrow Sectrum: •Penicillin G via injection •Penicillin V orally •Effective mainly against Gram +ve e.g.: Strep, Staph, Bacteriodes Moderate Spectrum: •Ampicillin •Amoxycillin •Effective against G+ve and G-ve, used to treat Urinary Tract infections, especially those that are caused by E. Coli Method of Action: The bond between C & N breaks, then C binds to Transpeptidase inhibiting its action by inhibiting cell wall synthesis and activating cell autolysis. Method of Resistance: The bacteria produce Beta-Lactamase or Penicillinase, which breaks the bond between the Carbon & the Nitrogen, attaching the Carbon to the Hydroxyl Group, then De-Carboxylation	•M, O, F (Narrow Spectrum): Methicillin (not used anymore): was used for Staph Aureus, which is now resistant → Methicillin Resistant Staph Aureus (MRSA) (NOT USED NOW FOR MRSA) Mec A (Resistance) Gene: 70% of Jordanian Population have it, 20% of inflammations Oxacillin & Flucloxacillin: used against Staph resistant to Penicillins/Ampicillin •Carbencillin &Piperacillin (Carboxypenicillins) (Narrow Spectrum): used to treat G-ve Pseudomonas •Monobactam "Aztreonam" (Narrow Spectrum): Only effective against G-ve Resistant Enteric Bacteria. It has one B-Lactam ring. •Amoxicillin + Calvulanic Acid "Augmentin" (Broad Spectrum): Beta-Lactamase Inhibitor, effective against G-ve Resistant Enteric Bacteria	6 Sulphur Ring Structure Administered IM or IV •1st Generation: Cephalexin, Cephradine (Broad Spectrum): similar to Penicillin & Amoxicillin, but causes less allergic reactions and has a wider spectrum. •2nd Generation: Cephoxitin, Cefuroxime (Broad Spectrum): Cephoxitin treats Anaerobic Bacteria Cefuroxime treats UTI •3rd Generation: Cefotriaxone, Cefotaxime (Mainly against G-ve Enteric): penetrate the CNS, treats meningitis & blood sepsis. Also treats some G+ve such as Strep. Pneumonia •4th Generation: Cefepime (Mainly against G-ve Enteric): mainly effective against G-ve bacteria which cause Upper Respiratory Tract Infection

occurs...inactivating the Beta-Lactam Ring.	•Carbapenems "Imepenem, Meropenem" (Broad Spectrum): highly resistant to most B-Lactamases including Extended Spectrum B-Lactamases (ESBL) Used for serious nosocomial infections & infections caused by G-ve Resistant Enteric bacteria (used as a last resort). Effective against G-ve Enteric Bacilli: Pseudomonas Aeruginosa, Acinetobacter Method of Action: Inhibits Penicillase/Beta-Lactam Method of Resistance: 1) Changes in the permiability of the cell membrane occur; this shuts down the pores which allow Penicillin to enter through. 2)Producing Penicillin-binding proteins which have a low affinity0 for Beta-Lactams Mnemonic: Chris Found Cancer's Cure? Oh Please! My, My! •Carbenicillin •Fluloxicillin •Calvulanic Acid	

Microbiology summary

	•**Carbapenem** •**Oxacillin** •**Piperacillin** •**Monobactam** •**Methicillin**	

Antimicrobial Drugs: Drugs that Inhibit the Synthesis of Essential Metabolite			
Sulfa-Drugs/Sulfonamide	**Cotrimoxazole (Sulfmethoxazole-Trimethoprim)**	**Anti-Tuberculosis Drugs**	**Metronidazole**
Bacteriostatic **Rarely Used Alone** **Mechanism of Action:** The structure is analogous to **PABA**, so it competes with it **BLOCKING FOLIC ACID SYNTHESIS** (which is essential for **nucleic acid synthesis**). **Mechanism of Resistance:** Possesion of a rapis, altered enzyme which is no longer inhibitable by Sulfonamide	**Broad Spectrum** Used to treat **UTI & RTI** **Mechanism of Action:** Combines Effects/Synergism (Sulfmethoxazole-Trimethoprim) **Mechanism of Resistance:** Bacteria develops resistance	**Mechanism of Action:** Inhibition of **Mycolic Acid** which is part of the Mycobacterium Cell Wall. (*Mycobacterium Tuberculosis*- 6 months of treatment)	**ONLY AVAILABLE DRUG THAT TREATS 2 CLASSES OF MICROORGANISMS: Protozoa (Amoeba) & Anaerobic Bacteria**

Microbiology summary

Inhibition of nucleic acid synthesis				
	Nalidixic Acid (Quinolone)	Floroquinolones	Nitrofurantoin	Rifamycin / Rifampin
Bactericidal / Bacteriostatic	Bactericidal	Bactericidal	Bacteriostatic	Bactericidal
Spectrum activity	Broad Spectrum	Broad Spectrum	Extended Spectrum	
Gram +ve / -ve	Mainly Gram –ve, with minor anti-gram positive activity.	More –ve than +ve	Gram –ve enteric bacteria (E-coli)	
Drugs		CLON Ciproflaxin, Levaflaxin, Oflaxin, Norflaxin.		
Mechanism of action	Inhibits DNA gyrase which is responsible for developing the double helix / transcription.	Inhibit DNA gyrase and interfere with tRNA transcription.	Damages DNA.	Bind to RNA polymerase transcription from DNA.
Side effect	gastrointestinal effects such as nausea, vomiting, and diarrhea, as well as headache		nausea, headache, and flatulence.	fever, gastrointestinal disturbances, rashes.

	and insomnia.			
Resistance	mutations at key sites in DNA gyrase can decrease the binding affinity.	Altered DNA gyrase mutation develops during treatment.	Resistance rarely develops.	Change in RNA polymerase β-subunit
USES	Only effective in urinary tract infection.	Intracellular pathogens, urinary tract, pneumonia and septicemia.	Used for treatment of urinary tract infection.	
Notes	Considered an anti-uropathogenic drug		-Not a Quinolone - Considered an anti-uropathogenic drug.	- Last resort for certain infections. - Should not be used as single drug.

ANTIMICROBIAL DRUGS: INHIBITION OF PROTIEN SYTHENSIS					
	Aminoglycosides	**Tetracycline**	**Chloramphenicol**	**Macrolides**	**Lincosamides**
Bactericidal / Bacteriostatic	Bactericidal	Bacteriostatic	Bacteriostatic can become bactericidal if concentration increases	Bacteriostatic	Bacteriostatic

Microbiology summary

Spectrum activity	Broad spectrum	Broad spectrum	Broad spectrum	Narrow (?)	Broad spectrum
Gram +ve / -ve	Mainly –ve	-ve& +ve		+ve (intracellular)	-ve& +ve
Used for Anaerobes?	No	No	No	No (Erythromycin) Yes (Clarithromycin, Azithromycin)	Yes
Administration	IV or IM				
Drugs	**(SGANNT)** **Streptomycin**, **Gentamycin**, **Amikacin**, **Neomycin**, **Netilmycin**, **Tobramycin**.	Doxycycline, Minocycline. -New one: Tigecycline (-ve)		Erythromycin, Clarithromycin, Azithromycin – orally long lasting 12 hours –	Clindamycin, Lincomycin
Mechanism of action	Irreversibly bind the 30s ribosome and freeze the 30s inhibition complex.	1- Accumulation in cytoplasmic membrane 2- Inhibit essential enzyme 3- Prevent attachment of amino acyl tRNA to 30s ribosome complex	Binding to the 50s ribosomal subunit and blocking the formation of the peptide bond	Binding to the 50s ribosome subunit, inhibit either peptidyl transferase activity or translocation of peptide to mRNA	

Microbiology summary

Side effect	Nephrotoxicity, Otoxicity, 8th cranial nerve –>hearing loss, blood level monitoring.	Overgrowth of yeast (Candida species)	Highly toxic on bone marrow, Anemia (1 in 1000)		Pseudomonas otitis serious bloody diarrhea – due to Clostridium Difficle –
Resistance	Production of acetylate, phosphorylate, adenylate enzyme	Develop of resistance by reduced active transport and pumping efflux			
Other	- used in cases of serious infection - chromosomal and plasmid resistance - contraindication in pregnancy causing neonatal deafness -used for blood sepsis	- Never give tetracycline to a present women or children under 8. - Doxycycline, Minocycline -------->Cholera , respiratory and genital infection, mycoplasma, chlamydia, Legionella infection.	- it has a small structure, so it is easily distributed in the blood stream and everywhere in the body including the spinal fluids in cases of meningitis - one of the best drugs that cover the intracellular organisms as it goes inside macrophages and monocyte. - React intracellular bacteria	-Large lactone ring structure ranged between 14-16 membered rings. - Relatively non-toxic drugs. -Respiratory infections, Gram +ve pneumonia, Diphtheria, streptococci, staphylococcal,	-used for bone infection, oral cavity, anaerobic infections. - used against staphylococcus and streptococci. Lincomycin (or any drug affecting anaerobic bacteria) → increases Clostridium Difficle (Anaerobic spore forming)

Microbiology summary

			- can be used in case of meningitis, septicemia, typhoid, fever.	Mycoplasma, Chlamydia, Legionella pneumonia. - Used mostly in respiratory infection.	Produces enterotoxins Causes: Bloody Diarrhea, Pseudomembranous colitis.

Gram positive bacilli				
	Corybacterium diphtheria	Bacillus Cereus	Bacillus Subtilis	Bacillus Anthraces
Morphology	Gram positive pleomorphic rods	Gram positive rods	Gram positive rods	Gram positive rods
Spore forming	X	√	√	√
Transmission	Respiratory droplets from a carrier. Highly infectious (mostly children)	Endospores in easily contaminated food (rice, meat, fish)	Wound infections, sepsis	Endospores 1- Cutaneous (human cutaneous anthrax) 2- Inhalation (hemorrhagic pneumonia and septicemia)

Microbiology summary

				3- Ingestion (bloody diarrhea)
Metabolism	Facultative Anaerobic	Aerobic	Aerobic	Facultative Anaerobic
Virulence	Thick gray coating Pseudo membrane forms in the pharynx which serves as a base from where it secrets its toxins	Heat stable enterotoxin		1- Unique protein capsule: antiphagocytic 2- Virulence depends on acquiring 2 plasmids. One carries the gene for the protein Capsule; the other for its exotoxin
Clinical	1- Severe sore throat 2- Pseudomembrane forms on pharynx 3- Liver necrosis 4- Myocarditis and neural involvement	Food poisoning: Nausea, vomiting, and diarrhea NO FEVER	Sepsis	Anthrax. 1- Cutaneous (95%): painless black vesicles (can be fatal if left untreated) 2- Pulmonary 3- GI: abdominal pain, vomiting, and bloody diarrhea
Treatment	1- Antitoxin 2- DTP vaccine (diphtheria toxoid) three doses 2,4,6 age months children	No need for antibiotic		Surgery and antibiotics
Diagnosis	1- Albert`s stain direct smear and throat culture (metachromatic granules) 2- Tellurite blood agar (selective for cornybacterilum because within the first 24-48 hours we are not able to differentiate between cornynebacterium diphtheria and	Culture specimen from suspected food source.		1- Culture specimens 2- Blood/ sputum culture or blood and chocolate agar

Microbiology summary

	haemophilus influenza cocci)			
Toxin	Diphtheria toxin obtained from a bacteriophase by lysogenic conversion (exotoxin)	Enterotoxins		Exotoxin: 3 proteins ((READ ONLY)) 1- Proteoactive antigen (PA) 2- Lethal factor (LF) 3- Edema factor (EF)
Notes	Corynebacteria ↙ ↘ <u>Normal flora</u> <u>Pathogenic</u> Of respiratory Diphteria Tract, could become Pathogenic in immune-deficient patients	Incubation period: 1- 24 hours Very rare invasive infections. Rarely fatal.	Found infectious in children/adults as part of the normal flora. Becomes pathogenic in immunocomprimised patients.	Causes intestinal fatal disease in animals - Human cutaneous anthrax…chronic lesions - Inhalation of pores causes hemorrhagic pneumonia septicemia… high mortality, war - Skin ulcers are rare

Microbiology summary

GRAM POSITIVE SPORE-FORMING BACILLI				
	Clostridium Tetani	**Clostridium perfingens**	**Clostridium betulinum**	**Clostridium defficle**
Reservoir	Soil	- Soil - GI tract of humans and mammals	- Soil - Canned food	- Intestine tract (normal flora) - Endospores found in hospitals and nursing homes (common nosocomial infection)
transmission	Endospores: introduced through wound	Endospores: introduced through wounds	Endospores (heat resistant) ((ingestion of contaminated food))	Fecal-oral route by ingestion of endospores (endoinfection). Nosocomial infection following long antibiotic usage.
Metabolism	Anaerobic	Anaerobic	Anaerobic	Anaerobic
Toxins	Tetanospasmin: Inhibits release of GABA and Glysine (both are inhibitory neurotransmitters) from nerve cells (CNS) resulting in sustained muscle contraction	Enterotoxins: - Hyaluronidase - Collagenase (bothare spreading factors) - Hemolysins Destroy RBCs by attacking their plasma membrane	1- Neurotoxin: inhibits release of Ach from peripheral nerves. 2- Toxin is not secreted, rather it`s released up on death of the bacterium (preformed toxin)	1- Toxin A Enterotoxin which causes fluid accumulation in the intestine, diarrhea 2- Toxin B Cytotoxic
Treatment	- Surgical debridement - Antibiotics - Tetanus vaccine	- Radial surgery (may require amputation) - Debridement of tissue - Hyperbark oxygen - Mixed antibiotics (NO VACCINE)	- Antitoxin - Hyperbaric oxygen - Supportive therapy (Incubation + ventilation assistance)	1- Metaredazole 2- Varomycin 3- Discontinue unnecessary antibiotics

Microbiology summary

Lab diagnosis	- Aspirated specimens from damaged tissues - Direct gram stain - Culture on anaerobic blood and chocolate agar	- Culture specimens (requires anaerobic conditions) - Aspirated fluid wound/ blood - Gram stain/ PCR detect toxins	- Gram stain - Culture (requires anaerobic conditions)	1- Immunoassay for C.difficle toxin (identification of toxins in stool specimens by immunological tests) 2- Less culture
Notes	- Heat stable/ labile - Highly fatal without treatment - LOCAL infection - Produced by vegetative cells which grow in necrotic tissue under anaerobic conditions - Symptoms appear remote from the infection site (but t multiplies locally) - Drumstick microscopic appearance	A common cause of food poisoning (enterotoxin) Incubation: 6-24 hours *** watery diarrhea and abdominal cramps, NO FEVER	- Heat stable exotoxins (destroyed at 20min/100 Celsius) - Botulism: clinical symptoms begin 8-36 hours after toxin ingestion - Difficult to detect toxin/ bacteria - Highly fatal	- Normal flora in 20% of people and 30% of hospitalized people - Common nosocomial infection following long antibiotic usage. - Change in PH/ disruption of intestinal flora enhances the growth of clostridium defficle and production of toxins.

Gram Negative Coccobacilli					
	Haemophilus influenza	Bordetella pertusis	Neisseria gonorrhea	Neisseria meningitis	Moroxella Catarrhalis
Reservoir and Transmission	- Man only (obligate human parasite) - Transmitted via respiratory route	- Man (highly contagious) - Transmitted via respiratory route (droplet infection/ close contact)	- Humans only (no immunity to repeated infections) - Sexually transmitted	- Nasopharynx of humans only Immunity can develop to particular strains/ strict human parasite - Spread by respiratory	Part of the normal respiratory flora (opportunistic pathogen)

Microbiology summary

				transmission	
Virulence	- Capsule: 6 types A-F; B is the most virulent - Endotoxin: Hemolysin	1) FHA (Filamentus hemagglutin): a pili rod which extends from the surface of B. Pertussis enabling it to bind ciliated epithelial cells of the bronchi 2)Pertussis toxin	Pili: for adherence to epithelial cells/ antiphagocytic (binds bacteria tightly to host cell, protecting it from phagocytosis) 2.IgA protease 3. Outer membrane proteins for adherence	1- LPS 2- IgA protease 3- Polysaccaride capsule (Serotypes A, B, C)	
Metabolism	Micro- aerophilic (aerotolerent anaerobe)	Aerobic	Facultative anaerobe	Facultative anaerobe	Aerobic
Clinical	Encapsulated H-influenza (localized and invasive infection) 1- Meningitis 2- Septicemia 3- Pneumonia 4- Acute epiglottis/ sore throat/ Otitis media/ sinusitis/ conjunctivitis	WHOOPING COUGH Catarrhal phase: patient is highly contagious A- Low grade fever, runny nose, mild cough B- Antibiotic susceptible during this stage 2- Paroxysmal phase A- whoop B- Increased number of lymphocytes in blood serum C- Antibiotics infective	Inflammation o mucosa resulting in Urethral/ vaginal discharge - Urethritis/ cervicitis/ salpengitis - Common reinfection	1- Respiratory tract infection (initially) 2- Sore throat 3- Septicemia 4- Meningitis	Respiratory tract infections
Treatment	1- HIB vaccine (infants > 2months) 2- Antibiotics	1- Erythromycin (during catharral stage)	- Antibiotic - NO vaccine	1- Protective vaccine available for all capsules except B	1- Antibiotic according to susceptibility

Microbiology summary

	HIB is resistant to B lactamases (it produces B lactamases so 2nd/ 3rd generation of cephalosporin (READ ONLY)	2- DPT vaccine (2,4,6 months)		2- Antibbiotics	2- NO vaccine
Lab diagnosis	1- Gram stain 2- Chocolate agar and blood agar containing X and V factors	1- PCR 2- ELISA serological test (studying plasma serum to look at the presence of antibodies)	1- Direct gram stain 2- Rapid cultures in blood/ chocolate agar 3- Selective medium (Thayer-Martin medium)	1- Direct gram stain 2- Rapid cultures in blood/ chocolate agar 3- Selective medium (Thayer-Martin medium)	1- Gram stain culture 2- Rapid cultures in blood/ chocolate agar 3- Biochemical testes (fermentation)
Notes	1- H influenza requires 2 factors for growth (both are found in blood) - X factor (Hematin) - V factor (NAD+) 2- Haemophilus stands for blood-loving 3- Grows around hemolytic staphylococci 4- Rapid autolysis outside the body (autolysins activation) 5- Originally part of the normal respiratory flora but is opportunistic 6- Children 6 months to 5 years	1- Highly risk groups: - Infants less than 1 year old - Adults (as immunity acquired from vaccine lean off … so higher fatality) 2- Incubation period is 7-10 days	Morphology: Kidney bean shaped with concave sides facing each other forming the appearance of a doughnut. - Oxidase/ Catalase positive - Very susceptible to environment factors (low/ high temperature and dryness) - Men more easily diagnosed than women	Morphology: Kidney bean shaped with concave sides facing each other forming the appearance of a doughnut. - Oxidase/ Catalase positive - Very susceptible to environment factors (low/ high temperature and dryness) - Children 6 months to 5 years are most susceptible	Resistant to Penicillin Not an intracellular organism like Neisseria

Microbiology summary

Enteric bacteria					
	enteropathogenic	enterotoxigenic	enterohemorrhogic	Klebsiella-enteric bacteria	Proteus providencia
reservoir	Found commonly in waste water / normal water /soil/vegetation	Fecal water contamination/vegetables/fresh food *it's an indicator of hygiene*	*Originate mainly fromintestines of cattle *ground meat/hamburger/diary products	Part of the normal flora but less common than E-coli (5% only)	Found in the intestines in percentages less than E-coli &klebsiella-enterobacter
transmission	Fecal-oral route	Fecal-oral route	Fecal-oral route	*hospital acquired *wound	*hospital *wound
Aerobes/anaerobes	Facultative anaerobe	Facultative anaerobe	Facultative anaerobe	Facultative anaerobe	Facultative anaerobe
Lactose fermentation	YES	YES	YES	YES	NO
catalase	+ve	+ve	+ve		
oxidase	-ve	-ve	-ve		
Opportunistic ?	pathogenic	pathogenic	pathogenic		

Microbiology summary

Virulence/pathogenesity/toxins		Heat-labile- heat stable – enterotoxins (these inhibit the reabsorption of the Na+ & stimulate secreting of CL- & HCO3 , water follows → traveler's diarrhea (self limited)	*Shiga- like toxin ;Vero toxin (their toxins inhibit protein synthesis in the epithelium which leads to intestinal death)	*Klebsiella—k-antigen *encapsulated k.pneumoniae(can't be bephagocytosed due to capsule also aid in adherence *enterobacter –slive layer	*Large number of Fimbriae *Endotoxin causes fever and shock when septicemia occurs
Clinical	*mild to chronic watery diarrhea , less vomiting	*self limited diarrhea called "traveler's diarrhea"	*bloody diarrhea *Haemolytic – uremic syndrome (HUS) Uremia and anemia (kidney failure)	*encapsulated k.pneumoniae – k-antigen causes nosocomial infections & pneumonia * UTI *wounds→sept	Causes renal stones *common cause of UTI & hospital-acquired infection *causes septicemia (wounds)

Microbiology summary

				icemia *Rarely causes meningitis	
Who does it infect ?	Infants (1-6 months)	Children more than adults		Common in hospitalized patients	Common in hospitalized patients
treatment	Antibiotics	NO antibiotics , it's self limited	antibiotics	antibiotics	
Diagnosis	*E-coli culture , red on Macconkey Agar indicates lactose positive. *Gram stain	*E-coli culture , red on Macconkey Agar indicates lactose positive. *Gram stain	*E-coli culture , red on Macconkey Agar indicates lactose positive. *Gram stain	*Cultured on Macconkey Agar , red indicates Lactose fermenter *Gram stain	*cultured on Macconkey Agar (transparentcolorless→Lactose non-fermenter) *Gram stain
Fatal ?	YES	NO	YES	YES	

Microbiology summary

Urease test					Positive . able to break downurea .urine is alkaline because of urease activity.
Invasive		NO		Invasive in immunocompromised patients	
Notes	In general E-coli normally resides in the colon without causing disease (it's part of the normal flora) BUT it can acquire virulence factors ,making it pathogenic . (Normal flora + virulence factor = Disease) In general E-coli causes : *common UTI (40-80 %) *septicemia /wounds *neonatal meningitis # the classification of E-coli depend on which virulence factors E-coli possesses . In general all three types possess pili , capsules (k-antigen) & flagella (H-antigen)			Klebsiella and enterobacter are 2 different species and we can ONLY differentiate between them by Genetic Molecular Test	

Microbiology summary

VIBRIO CHOLERA, BRUCELLA, CAMPYLOBACTER, HELICOBACTER, ACINETOBACTER					
	Vibrio Cholera	**Brucella**	**Campylobacter**	**Helicobacter**	**Acinetobacter**
Virulence/ Pathogenicity/ Toxins	- Intensive motility by long flagellum (**H antigen**) - produce **cholera toxin- enterotoxin** which look like ETEC - **heat labile** - attached to mucosa of intestine by fimbrae	- produce endotoxins - highly infectious - enter through GI, skin abrasions, eye or via inhalation / droplet. **Then it's phagocytosed and lives intracellularly** (in macrophages) *release of endotoxins from macrophages causing weakness, sweating…	- motile (**bipolar flagella**). This motility is responsible for clearance from intestine	- **release urease** which convert urea to CO_2/bicarbonate & ammonia → <u>neutralize stomach acidity</u> & protects its colonies - it produces gastritis or peptic ulcers in the stomach or duodenum by using **multi uni-**	- not so virulent as it's not motile - multiple acquired mechanism antibiotic resistance

Microbiology summary

				polar flagella for adherence then producing urease to neutralize the acidity of the stomach	
Clinical	- **VERY SEVERE watery diarrhea** (rice water stool) ; severe dehydration - cause blood acidosis if patient is not	- caused localized infection in animal's reproductive organ, sepsis, and abortion in cows (induced abortion) - Brucellosis/Malta fever : **intermittent fever** - elevated	- cause abortion & enteritis in sheep & cattle - multiply in small intestine, invade the epithelium →produce mild inflammation causing **watery-bloody diarrhea** (moderate to mild diarrhea).	- **gastritis/ peptic ulcers in stomach or duodenum** - nausea, vomiting - Persistance of ulcers: increased risks of stomach cancers and lymphomas	- pneumonia - sepsis - meningitis

	rehydrated → hypervolemic shock → lead to death within hours (24 hours) - vomiting, muscle cramp (leg)	in night, slowly return to normal by morning, fatigue, joint & bone pain, sweating, headache, GI symptoms, septicemia, meningitis, chronic disease with CNS complication, vomiting, diarrhea, abdominal pain, hepatosplenomegaly	- Few fecal leukocytes found in feces - cause sepsis in children - fever, abdominal pain, nausea, headache & muscle pain - Complication : arthritis, Gullian- Barre Syndrome		
Who does it affect ?					Hospitalized or immunedeficient patient
PH	- Alkaline (			- Acidic (pH	

Microbiology summary

	pH 8-9) - infect intestine			2-4) - Infect stomach	
Morphology	- Comma shaped like a hilal with a tail	- Coccobacilli	- Thin, curved, motile * Found in cluster where as V.Cholera found in single cluster	- Spiral shaped with multiple flagella	Pleomorphic (change their appearance to cocci/diploid like Neisseria
Notes	- Not part of Enterococcae. It belonged to family of Vibriocacea -Like Salmonella Typhi / Paratyphi, it only	- Lives **INTRACELLULARLY** - it's a special group that belong to a different family (Brucellacae) - FUO (fever of unknown origin) - Slow growing * Difference between enteric	- Optimal growth (42°C)(animals ?) *Campylobacter can't be clinically distinguish from Salmonella/Shigella infection BUT abdominal pain tend to be more severe in	- Only pathogenic in human - Up to 10% in children, 80% in adults can have evidence of H.Pylori colonization (mild infection) -Common	- Survive for extended period on surface, so they're easily transmitted - The commensal bacteria are of low virulence (opportunistic pathogen)

	infects human & is associated with intestinal inflammation -Survive in any kind of water for a long time - Produce “Cholera Toxin” which is also produced by ETEC, *the difference is not by the type of toxin	fever caused by Salmonella Typhi/Paratyphi and Brucella is that undulating(caused by Brucella)	Campylobacter infection . Both infection lead to watery-bloody diarrhea *Campylobacter also produce ulceration in intestine & stomach and this is associated with the released of damaged blood vessels & released of blood filled stool	recurrence within few weeks to months *Special : it’s the only organism that can survive in stomach’s acidity - Availability of certain condition is necessary for the disease to progress	- Can be part of skin & nasal normal flora

	produced but by the amount of toxin released. ** V.Cholera release 10-100 times more than toxin released by ETEC -Antibiotics can be given only to reduce the excretion of the organism in stool as well as to				

	prevent dissemination of the organism in the community				
Reservoir	- Contaminated water - Human intestine (only human are infected)	-**Brucella Abortus** : cause abortion in cows - **Brucella Melitensis** : infects goat/sheep	-Widely spread in small animals, dogs, cats, birds -It's primarily an animal pathogens	- Mucus lining the stomach & duodenum causing chronic inflammation like gastritis and ulcers	- Commonly found in water, moist hospital environment (found in skin, urine, wound, sputum)
Transmission	Fecal-oral route through contaminated water, fresh food	By ingestion of infected food particles (diary product), direct contact with an infected animal/inhalation	Ingestion of contaminated chicken meat, milk, fresh food, water	Close personal contact	Nosocomial infection in immunodeficient & ICU patient

Microbiology summary

Aerobic/Anaerobic	Aerobic	Microaerophilic	Microaerophilic	Microaerophilic	Aerobic
Lactose Fermentation	-ve				-ve
Catalase					+ve
Oxidase	+ve				-ve
Opportunistic?			Yes		Yes
Treatment	-Rapid replacement fluid (IV) & electrolyte with antibiotics - Doxycyclin : to shorten illness duration - control through public health	- 6-8 weeks with antimicrobial drug - Vaccine mainly used in animals	- **Self-limiting**; no need for anti-microbial drugs. *The patients might suffer of diarrhea for 2-3 days but no need for anti microbial drugs - No vaccine available	- Can be successfully eradicated (95%) using a combination of certain antibiotics & medicine that suppress stomach acid production (3 antibiotics) - No vaccine	-The only drug available is Colistin but it's very toxic & might produce renal failure - No vaccine

	sanitation measurem ent & human vaccine - Mild : oral rehydratio n				
Diagnosis	- Feces culture and selective **TCBS (Thiosulfat e- Citrate Bile Salts Sucrose Medium**), where is recognize as yellow colonies on the surface of the	- from blood, CSF, bone marrow culture in chronic brucellosis (1-4 weeks culture incubation period), agglutination test, specific antibodies (IgM, IgG) - Brucella skin test indicate exposure only	- grow slowly over 3-6 days in vitro culture - stool culture selective campylobacter media including **3 antibiotics**, biochemical test - direct detection of bacteria by PCR	- **Urea breath test** - **culture biopsy stomach** - **selective medium with 3 antibiotics** - **incubation 37°C, 4-6 days** - **Serological test** * H.Pylori antibodies	

Microbiology summary

	medium (16 hours) - Gram stain - Specific motility test - Anti-sera against cholera	(not so useful)		not significant for clinical diagnose alone	
Fatal ?	Yes				
Urease Test				+ve	
Invasive ?	No		To some extent		
Incubation Period	8-48 hours	-Generally 1-2 months/1-6 weeks	2-5 days in human	4-6 days (37°C)	
Healthy Carrier	Yes (10-50%)			Yeah, sort of	Assymptomatic skin & nasopharyngeal carriage
Serotype	V.Cholera 01: Type El-Tor	- Brucella Abortus - Brucella	Many. Most infection in human caused		

Microbiology summary

		Melitensis	by : - C. Jejuni -C. Infantis		
Resistance					Develop rapid resistance to most used antibiotics & is also multi drug resistance
Survive under harsh conditions ?	It's salt tolerance. (survive in any types of water)				Yes (It can survive on moist and dry surfaces, including in a hospital environment.)

Microbiology summary

PSEUDOMONAS AERUGINOSA & ENTERIC BACTERIA (GRAM NEGATIVE BACILLI) : SHIGELLA, SALMONELLA ENTERICA/TYPHI				
	Pseudomonas Aeruginosa	**Shigella Dysenteriae**	**Salmonella: 4 Disease States in humans: Diarrhea (Gastroenteritits), Typhoid Fever, Sepsis, Carrier State**	
			Salmonella Enterica (Non-Typhoidal Diarrhea/Gastroenteritis)	**Salmonella Typhi (Typhoid/ Enteric Fever)**
Reservoir	•May be found in the human intestine under normal conditions, but not as commonly as the Coliform group. •Colonize in the URT &intestines •Soil/Water/Plants/Animals	•Found in the intestines of humans, BUT is NEVER part of the normal flora, it is always a PATHOGEN.	•GI tracts of animals (mostly birds, farm animals: chicken/cows) •Common in nature (water/soil cotaminated with animal feces) • NEVER part of the normal flora, it is always a PATHOGEN.	•Carried ONLY by humans •Only a few bacterial cells are needed to inititate an infection •After invading the intstinal epithelium, it invades the regional lymph nodes, and seeds in multiple organ systems (sepsis). *Note: it's a facultatuve intracellular parasite
Transmission	•Medical devices (Endotracheal tubes, Urinary Catheters) •Hands of Health Care Workers	•Fecal-Oral route via fecally contaminated water •Hand-to-Hand contact	•**<u>Food Borne Disease:</u>** *Contamination of fresh food/water with animal feces *Raw eggs/chicken/meat *Fresh Unpasteurized dairy products •**<u>Zoonotic Disease:</u>** Infectious disease that can be transmitted from animals to humans	Fecal-Oral route via contaminated food and water

Microbiology summary

Aerobic/Anaerobic	Aerobe/Facultative Anaerobe	Facultative Anaerobe	Facultative Anaerobe	Facultative Anaerobe
Lactose Fermentation	No	No *Note: Shigella is also immotile (lacks flagella). These two characteristics can be used to distinguish Shigella from E.Coli, which is motile and a lactose fermentor.	No	No
Oxidase	Positive	Negative (All Enterobacteriaceae are Oxidase Negative) *Note: Enterobacteriaceae is a family within Enteric Bacteria		
Opportunistic	Yes (won't affect a healthy person)	Yes	Yes	Yes
Virulence/ Pathogenicity/ Toxins	•Releases may enzymes (Collagenase, Elastase, Hemolysin, Fibrinolysin,DNAase) •Exotoxins •**Pyocyanin**: A dye which is an antibiotic-like substance that can inhibit the growth of other Coliform & kill them. (Survival on the expense of other organisms)	•**Shiga Toxins: secreted during the multiplication of the organism inside the small intestines.** •Endo/Enterotoxins released in the intestines (like EHEC): Inhibits protein synthesis and kills intestinal epithelial cells •Neurocytotoxin: affects the CNS	•**O & H antigens: O for the protection against antibodies, H for motility** (These antigens have specific antibodies) •**Cholera-like Toxin** •Endotoxin/LPS	•**O & H antigens: O for the protection against antibodies, H for motility** (These antigens have specific antibodies) •**Vi-layer** (in Typhi only,not in Para-Typhi)

Microbiology summary

Fatility	Fatal	Fatal without treatment	Fatal in untreated/immunocompromised patients.	Fatal
Invasiveness	Invasive	Might reach the blood stream of immunocompromised patients	Less invasive than Salmonella Typhi	Highly invasive
Incubation Period	28 Hours	1-2 Days	8-24 Hours	1-3 Weeks
Healthy Carriers	May be found in the human intestine under normal conditions, but not as commonly as the Coliform group.	No	Yes	Yes, mostly women, in the Gallbladder for a short period or life-long
Serotypes	There are 20 species of Pseudomonas that cause infection, P.Aeruginosa is one of them.	Shigella Dysenteriae, Shigella Sonnei, Shigella Boydii	There are 2,000 serotypes of Salmonella	Typhi and Paratyphi A, B, & C
Notes	•Not part of Enteric Bacteria •Produces a green flourescent pigment, and a blue pigment (pyocyanin) •Very resistant to antibiotics (Even more resistant than the Coliform group)	Cannot survive for more than 30 minutes outside the human body, so it should be cultured immediately	Self-limiting in healthy persons, but mainly infects immunocompromised patients (very dangerous)	•Typhi is more dangerous than Paratyphi due to the presence of Vi slime layers within its cell wall. Paratyphi A, B, & C aren't associated with this virulence factor •The bacteria is phagocytosed by monocytes, and therefore can survive intracellularly as a **Facultative Intracellular Parasite**

Microbiology summary

Survival Under Harsh Conditions	Resistant to alcohol used in aseptic procedures, detergent (difficult to penetrate the cell wall)	Susceptible to acidity, dryness, and temperature		
Who it Affects	Hospitalized/ Immunocompromised Patients (P. Aeruginosa is a hospital-acquired infection)	Only Humans	Humans and Animals	Only Humans
Clinical Aspects	BE PSEUDO B: Burn-Wound Infections E: Endocarditis P:Pneumonia S: Sepsis E: External Otitis Media U: Urinary Tract Infection (from contaminated Catheters) D: Diabetic Myelitis O: (none)	•Purulent Bloody Diarrhea (Bloody diarreha full of pus a.k.a. bacillary dysentery) •Severe intestinal Necrosis •Abdominal Pain, Fever •Rare Septicemia •Minimized Vomiting (it is an intestinal disease)	•Large number of Salmonella cells can cause diarrhea •Bloody, mostly watery diarrhea (mild to severe) •Vomiting •Rarely causes Septicemia or Meningitis	**•High Continuous Fever (40-41°C)** •Bloody Diarrhea •Constipation •Intestinal Perforation Vomiting **•Hepatosplenomegaly** •Septicemia •Meningitis •Osteomyelitis •UTI
Lab Diagnosis	Produces a green	•**Doesn't produce**	•**Produces H2S** upon	•**Produces H2S** upon

	flourescent pigment, a blue pigment (pyocyanin), and a sweet smell	**H2S** upon rapid culture with the **Salmonella-Shigella Agar** (rapid feces culture/rectal swabs) •**Transparent colonies** on the **Hektoen-Enteric Agar** •CANNOT be cultured on the McConkey Agar	rapid culture with the **Salmonella-Shigella Agar** (culture of feces or food) •**Transparent colonies** on the **Hektoen-Enteric Agar** •**Widal Test** of specific antibodies against **O & H antigens**	rapid culture with the **Salmonella-Shigella Agar** (culture of feces, urine, blood, cerebrospinal fluid) •**Transparent colonies** on the **Hektoen-Enteric Agar** •**Widal Test** of specific antibodies against **O & H antigens**
		•Prevention/control through hygeine and sanitation in restaurants,slaughter houses, and food handlers.		
Treatment	Complicated treatment, the antibiotics are chosen depending on what the P. Aeruginosa caused.	Unlike Salmonella Gastroenteritis, there may be severe necrotizing inflammatory reactions, so it is	The diarrhea of Salmonella Gastroenteritis is usually self-limiting, so we just administer fluid and electrolyte replacement.	Ciprofloxacin & Ceftriaxone Vacccine is available

	*Antipseudomonal antibiotics include: -Antipseudomonal Penicllins -3rd Gen. Cephalosporins: Ceftazidime, Cefoperazone -4th Gen Cephalosporin: Cefepime -Carbapenems -Aztreonam -Ciprofloxacin -Aminoglycosides	recommended to administer antibiotics		

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Microbiology summary