Review of Material Covered On First Midterm

		Note: Numbers in left hand column correspond to slide numbers on slide show titled "Review for First Midterm". Make sure you download this from Blackboard as well and follow along.
2	Where do we find micro-organisms?	- Everywhere (environment, animals, plants…) - They aren't all bad - Intestinal flora - Ex of micro-organism? bacteria
2	What is immunology?	Study of our protection from foreign macromolecules or invading species and our response to them.
2	Classes of micro-organisms	- viruses >> grow only in living cells - mycoplasma >> grow on non-living media - bacteria >> no separate nucleus - parasites - Most complicated? bacteria >> separate nuclei
2	Are all bacteria bad?	- No. - Resident vs. Transient - Normal gut flora >> protects a person from invasive organisms and helps with metabolism (esp. of vit K) - antibiotic therapy >> displaces normal flora >> secondary deficiencies - No normal flora at birth! - where should there be no bacteria? >> eyes, lungs, brain, blood, CNS, urine - Normal flora >> balanced eco-system - Infection >> one species becomes dominant or bacteria invades area which is meant to be sterile
3	Basic structures: viruses	
4	What protects us from the bad guys?	
5	Mechanical Barriers	- Skin - pH **(acidic or basic)** - possible for organisms on skin to cause infection if they penetrate the skin (ex: wound/ needle)
5	Adherence	- In order for a bacteria to infect a host, it must bind to a host cell
5	Phagocytosis* 6	- macrophages and blood neutrophils engulf, kill and ingest bacteria - some bacteria (ex: Strep pneumonia) form a protective polysaccharide capsule called an antiphagocytic coat which prevents phagocytosis.
5	Antibodies (humoral)	- people form antibodies against antigens - can act as an opsonin or directly kill antibody

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6	Opsinization	
5	Complement	- circulating proteins that fight against invasive bacteria - 2 roles: 1. opsinization 2. forms the membrane attack complex >> recognize sugars on the wall, punches a hole in it and lets the insides leak out. Basically kills infected cells.
5	Cell mediated Immunity	- T-cells. Secondary response.
8	Pathogenicity	a micro-organism's ability to produce disease
8	Virulence	relative capacity to cause damage (ex: the degree of pathogenicity). Basically how severe a disease it can cause
8	Opportunistic	Does not normally cause disease, but can do so when defence mechanicsm are compromised.
8	Micro-organism	organisms invisible to the named eye. The majority are harmless nd many are helpful. A small portion produce harmful effects on humans and animals.
8	Clinical Infection and Nosocomial infection	Clinical >> apparent symptoms. symptomatic opposed to asymptomatic. Subclinical >> asymptomatic Nosocomial >> hospital acquired.
9	Pathogenicity of infectious diseases	- When a microbe enters the body >> 2 responses: 1. microorganism tries to multiply, invade and cause disease, and 2. host tries to prevent effectiveness of the microorganism - Whether the host or the microbe wins depends on many factors, some of which include: - Transmission (2 most common = inhalation and ingestion) but also a break in protective barriers incl. skin, etc. - Pathogenicity >> capacity of microbe to effect the host depending on 1. invasiveness and 2. toxigenicity. - Invasiveness >> microorganisms overcome host defence by 1. adherence, persistence and avoidance of the immune system. - Toxigenicity >> ability to produce toxins (either exotoxins or endotoxins).
11	What is immunity	- Our body's ability to resist infection in order to prevent us from disease by generating an "immune response" by immunization, previous infection or by noon-immunological factors. - 2 Types: innate and adaptive

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11	Innate (non-specific) immunity	- protects agains ANY invader; does not discriminate - skin >> mechanical barrier to microbes; acidic pH; sebaceous secretions are bactericidal; low temperature discourages microbe growth - mucous membrane >> mechanical barriers; ex: cilia in resp. tract, lysozomes in tears; vaginal, gastric and urinary pH - iron-binding proteins >> prevent growth of iron-dependent bacteria; ex: transferrin, lactoferrin. - phagocytosis - complement
11	Adaptive (specific) immunity	- directed against specific type of invading micro-organism; dependent on past exposure - humoral and CMI
13	Primary Immune Response:	- antibody production triggered upon first introduction of antigen - Ab detected after ~10 days
13	Secondary Response	- basis for immunization - occurs at 2nd, 3rd, 4th etc. introduction of antigen. - rapid Ab increase, slow decrease - Ab detected after 2-3 days - boosters >> maximize Ab levels
13	Latent period	- Period between exposure to antigen and detection of antibody - Decreases during secondary response - Immunization is key
20	Resolution	ability to distinguish two closely located objects as separate, distinct entities
20	Make a smear	- a thin film of specimen is placed onto a clean microscope slide and air dried
20	Fix dried smear by heat	- the dried smear is fixed by heat to make micro-organisms stick to the glass slide
20	Stain with desired dye	- stain with one or more dies prior to viewing with the microscope

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20	Selective vs. Differential	- Selective >> one dye; observe patterns/ colonies etc. Media is designed to enhance growth of one micro-organism while inhibiting growth of others. - Differential >> usually 2 dyes; shows the difference between micro-organisms. Differentiates based on nutritional requirements ex: pink >> lactose fermenting. clear >> non-lactose fermenting - S/D >> very useful in clinical settings (ex: MacConkey) - enrichment media >> favours growth of one species by meeting specific nutrition requirements - tissue culture media >> for cultivating viruses >> derived from plant or animal cells
		Note: The powerpoint continues with more review for the first midterm. Finish going through the powerpoint before returning to this document for the second midterm review.

Review of Material Covered on Second Midterm:

Lecture Five: Gram Positive and Gram Negative Bacilli:

Gram Positive >> spore formers and non-spore formers	- **spore formers:** cause disease by releasing potent exotoxins. - Differ absed on their ability to grow in the presence of oxygen. - Oxygen lovers: Bacillus “aerobic” - Oxygen haters: Clostridium “anaerobic”
Aerobic: Bacillus >> anthrasis and cereus	- **anthrasis:** causes anthrax >> effects herbivores (sheep/ cows) - only bacteria w. capsule made of protein - spores are stable/ resistant to heat, drying, UV and disinfectants. - human exposure through contact with infected animals/ soil - biological terrorism/ warfare - exotoxin composed of 3 separate proteins: edema factor. protective antigen and lethal factor. Separately, proteins are not toxic but together they are lethal. - **cereus:** motile, non-encapsulated, resistant to penicillin. - spores in food >> food poisoning >> enterotoxin responsible for food poisoning. - 2 types of enterotoxins: heat-labile and heat-stable

Anaerobic: Clostridium >> botulinum, tetani, perfringens, difficile	- **Botulinum:** produces a lethal neurotoxin >> neurotoxin blocks release of Ach in ANS >> flaccid muscle paralysis results. - Note: bilateral cranial nerve palsies, double vision, difficulty swallowing, general muscle weakness and respiratory paralysis - Associated w/ eating smoked fish/ home canned vegetables - "Infant botulinum" >> "floppy baby" >> fresh honey containing spores - **Tetani:** causes tetanus - rusty nail containing spores enters the skin >> wound creates an anaerobic environment >> spores germinate >> causes sustained contraction of skeletal muscle - exotoxin called >> "tetanospasmin" - lock jaw >> "trismus" >> high mortality rate **Note: FALSE >> stepping on a rusty nail will cause tetanus. For tetanus, spores are required to make the tetanus exotoxin called tetanospasmin **Note: be able to compare and contrast tetanus and botulinum as they are opposites. - **Perfringens:** causes gas gangrene >> spores mature in anaerobic conditions and produce gas - affects soldiers - 2 classes of infection: celulitis (wound infection) and myonecrosis - celulitis >> bacteria causes local tissue damage >> palpitation reveals moist, spongy, crackling consistency to skin - Note: pockets of gas called "crepitus" - myonecrosis >> muscle damage >> black fluid excreted from the skin - treatment for perfringens: hyperbaric oxygen, antibiotics (including penicillin), and removal of necrotic tissue - **Difficile:** 2 toxins >> Toxin A causess diarrhea and Toxin B attacks colonic cells - difficult to treat with antibiotics - patients should be taking a probiotic to replace empty space in flora and promote healthily immune system during antibiotic use
Non-spore formers: listeria and corynebacterium	- **Listeria:** Franco's bacteria!! - symptoms of pregnancy mask symptoms of listeria. pregnant ladies should avoid soft cheeses and deli meat - crosses the 3 protective barriers: blood-brain, gastrointestinal, fetoplacental - "facultative intracellular organism" >> can grow inside or outside a living cell - Psychrophile >> survives at refrigerator temperatures - **Corynebacterium diphtheria:** damages heart and neural cells - Exotoxin contains 2 subunits: A and B. - Subunit B allows A to enter, and subunit A blocks protein synthesis. **Note: don't confuse subunit A and B with toxin A and B from C. difficile.
Gram Negative Bacilli	- Gram Negative Bacilli are called the "Enterics" >> cause problems in the gastrointestinal tract - enterics are part of the normal intestinal flora and can also cause gastrointestinal disease >> "opportunistic" - Try to remember the unique characteristics of each enteric - Gram Negative Bacilli are classified based on their ability to ferment lactose as well as their surface antigens: - O = outer layer - K = capsule covering O-antigen - H = flagellar subunit (only in motile bacteria)

Enterobacteriaceae:	- **Salmonella:** unable to ferment lactose - infections of man: either “enterocolytic” (tummy problems), or “Enteric fever” (either typhi or paratyphi) - poultry, eggs, meat, milk - thrives on antibiotics therefore do not use antibiotics in treatment (we don’t know why) - **Shingella:** small dose of organism causes disease - no vaccines available - bloody diarrhea - **E-coli:** ferments lactose - 2 strains: ETEC and EHEC - ETEC: responsible for traveller’s diarrhea and infantile diarrhea in developing countries - EHEC (E-coli O157:H7) causes HUS “Hemolytic uremic syndrome) >> RBC destruction, kidney failure, can be fatal. AKA Hamburger disease. - **Enterobacter spp. (Cronobacter):** affects low birth weight babies, associated with powdered infant formula - **Other Enterobacteriaceae:** mostly nosocomial (begin in hospital).
Vibrionaceae:	- **Vibrio cholera:** causes cholera, a gastrointestinal illness - enterotoxin disturbs electrolyte balance leading to massive amounts of diarrhea (10-15L per day) - leads to severe dehydration and death - common in countries w/ poor water sanitization (think S.E Asia and parts of Africa) - **Campylobacter:** (do not confuse w/ cronobacter) - most common cause of the flu - microaerophillic (very sensitive to O2 levels) - 2 strains: C. jejuni and C. coli - part of normal flora in birds and domestic animals
Pseudomonas:	-grape smell/ purple gauze >> always pseudomonas - 2 species to know: - **Pseudomomas aeruginosa:** respiratory pathogen in patients w/ cystic fibrosis - hard to get rid of bc it forms a capsule once in the respiratory tract - also affects burn patients - **Pseudomonas cepacia:** common contaminant of saline solutions and water because it is able to multiply in low nutrient environment
Haemophilus influenzae:	- even though it is classified as an enteric, it is a respiratory pathogen (affects the resp. system) - exists in normal nasopharyngeal flora - significant pathogen in 2 situations: patients with invasive infections and children under 5 years old - remember: <5yrs old. - vaccine available
Legionella	- grows in water (commonly found on shower heads, air conditioning units, water tanks…) - exposure is by aerosol ** NOTE for exam: person to person transmission does not occur w/ Legionella - causes Legionaries disease
Helicobacteriaceae:	- only enteric that can survive stomach pH. - causes stomach ulcers

Alcaligenaceae: Bordetella pertussis	- causes Bordetella pertussis (whooping cough) - violent cough - 4 different virulence factors

Lecture 6:

- This lecture is pretty strait forward if you remember what we talked about in review. The key topics to review include the following:
    - Mycobacteria (tuberculosis, leprosy)
    - Other bacteria (spirochetes, mycoplasmas, Chlymydia, Mycoplasmas, Fungal Infections)
- For fungal infections, review the levels of infection (superficial >> cutaneous >> subcutaneous >> systemic).
- Remember to go over what we discussed in review as well as what you found to be most important.

Lecture 7: Parasitism

Parasitism: The innate and obligatory symbiotic relationship between two organisms of different species. The parasite depends on its host either long term or short term:

- long term parasite (example >> tapeworm)
- short term parasite (example >> mosquito)

Intermediate host: secondary host for the organism. It lives here and then moves on. Franco uses a condo in florida as an example.

Vector: carries the parasite from one host to the next (ex: mosquito)

Dead end host: The parasite ends up here “by accident”, infecting the host but then sacrificing itself. Humans are a dead-end host for a lot of microorganisms.

How successful a parasite is depends on:

- prevalence in host
- number of host species available
- geographic range
- number of offspring
- available routes of transmission

Protazoa (single-celled parasites):

1. **Giardia lamblia** (excess lipids in stool; high prevalence in children, travellers and immunocompromised; challenge to vaccinate against because antigens change throughout it’s lifecycle; control = water treatment)

2. **Trichomonas vaginalis** (unlike chlymidia there are no stages to remember; most common STI world-wide; symptoms include itching, foul-smell and frothy vaginal discharge; increases patient's susceptibility to cervical caner/ HIV; infection during pregnancy can lead to still birth and premature labour).

3. **Entamoeba histolytica** (common in developing, tropical countries; transmission though faecal-oral route, person to person, contaminated water, raw produce, food handlers and flies; largely related to poor hygiene/ sanitization; causes bloody, mucoid diarrhea; prevention by washing fruits and vegetables and avoiding tap water).

4. **Toxoplasma gondii** (cats! pregnant women should not change cat litter and should wash their hands after gardening; cats are the only definitive hosts; transmission through oocyte-containing cat faeces as well as raw meat; important that cats don't eat raw meat or catch wild prey).

5. **Plasmodium spp.** (Group of 4 different protozoa causing different types of malaria; plasmodium resides in RBCs and causes malaria. We see symptoms when RBCs are destroyed; No symptoms when Plasmodium is inside a RBC; transmission is by anopheline mosquitos; african people and people w/ sickle celled anaemia can't get malaria; symptoms include spiking fever and chills; remember the vector must break the skin in order for infection to occur; prevention includes prophylaxis, long sleeved clothing, bed netting and insect repellent). Protazoa causing malaria include:
    - vivax
    - malariae
    - faciparum
    - ovale

6. Cryptospuridium species: (Causes cryptosporidiosis; most common symptom is watery diarrhea; other symptoms include nausea and anorexia; life cycle includes sexual and asexual phases; no treatment available; mode of transmission is usually drinking water, pools and waterparks as well as person to person transmission, auto infection andzootonic transmission via cattle and calve faeces for some species of Cryptospuridium).

7. Cyclospora cayetanensis: (endemic in countries including Nepal, Haiti, Peru and Guatamala; low infectious dose; most common mode of transmission is water/ food; Food contamination can be either direct or indirect).
- Direct (infected pickers/ sorters/ inspectors/ food handlers)
- Indirect (contaminated water use for irrigation, mixing pesticides, washing equipment and hand washing).

Metazoa (multi-celled parasites)

1. **Nematodes (roundworms)**

    - **Enterobius vermincularis (pinworm):** (highest incidence in school aged-children; affects 50% of children in N. America; more of a nuisance than a health proble,;

transmission through faecal-oral route/ ingested eggs; leads to itching of perianal area; scotch-tape test)

- **Trichinella spiralis:** (small round worm; transmission through ingested larvae in raw/ poorly cooked meat)

- **Ascaris lumbricoides:** (very large intestinal nematode; transmission through ingestion of eggs in soil, fruits, vegetables and water)

- **Anisakis simplex (Whale worm/ Herringworm):** (most cases linked to home-prepared sushi and sashimi; definitive hosts include dolphins, whales and proposes; first intermediate hosts include marine crustaceans; second intermediate hosts include salmon, mackerel, cod, herring, tuna and squid; humans are dead-end host; diagnosis is very difficult b/c no eggs present in stool; control includes inspection of fillets at processing plant, candling on a light table to reveal larvae and cooking/ freezing fish).

2. Platyhelminths (flatworms)
- Remember key points from your notes!

- **sestodes (tapeworms)**

    - **Diphyllocathrium lactum (fish tapeworm) 3-10m long**

    - **Taenia saginata (beef tapeworm) 4-5m long; associated w/ weight loss**

    - **Taenia solium (pork tapeworm) 2-7m long**

- **Trematodes (flukes)**

    - **Schistosoma spp (blood flukes):** free swimming larvae in fresh water which penetrate skin and develop near bladder/ intestine.

****Good luck everyone and remember what I have here is just a summary of things I find important. Please use this as one study tool but don't forget about your course pack, slides and class notes!****

Review of Remaining Material to be Covered on Final Exam

****Remember, your final is cumulative but a large percentage of the questions will be based on the material from the last three lectures****

Lectures 8 and 9: Viruses

Basic Components of Viruses:
- nucleic genetic core >> contains viral genetic information
- capsid (protein coat) with antigens
- antigen = variable

Detection of Viruses:
- Either by identification of the virus itself
- Or by identification of the immune response (antibodies)

Identification of the virus itself:
- visualization by electron microscopy
- cell culture
  - cytopathic effect (destroys host)
  - heagglutination (RBCs)
  - immunoflourescence (doesn't destroy host)
- immunoclogical methods (direct ELISA)

Identification of the immune response (antibodies):
- immunity test (antibody presence)
- diagnostic test (titre of Ab)
- note >> time lag for development of antibodies with this method

Replication of viruses:
1. Adsorption
2. Penetration and Uncoating
3. Synthesis of Nucleic Acid and Protein (Separately)
4. Assembly and Maturation
5. Release by Lysis or Budding

** NOTE: most antivirals act on replication of nucleic acid.

Properties of Viruses:
- grow only in living cells
- contain either DNA or RNA never both
- protein/ nucleic acid divide separately and then join together >> forming virus
- TRY TO REMEMBER which viruses have and don't have vaccines

Viruses Causing Disease in Humans:

1. Respiratory Viruses

1.1 Influenza
- 2 Major types:
- Type A >> major epidemics
- Type B >> minor epidemics
- Recombination >> variability >> influenza pandemics

- highest risk = aged under 25 and over 65

1.2 ParaInfluenza
- Leads to Croup
- children >2 years old

1.3 Respiratory Syncytial Virus
- children <2 years

1.4 Rhinovirus
- common cold
- >100 serotypes
- no cross immunity
- repeated infections

1.5 Adenovirus
- respiratory virus
- army >> vaccinate

2. Enteric Viruses
- grow in GI tract
- cause problems elsewhere
- migrate via viremia
- infect lymphoid/ GI cells
- 95% inapparent

2.1 Poliovirus
- Types 1,2,3
- infects CNS >> paralysis
- causes poliomyelitis
- 2 vaccines:
- Salk >> killed (inactivated); IgA; no immunity; immunocompromised patients
- Sabin >> live attenuated; IgA and IgG; creates immunity in GI tract; administered orally; prevents spreading of virus; not recommended for immunocompromised patients

2.2 Coxackievirus
- leads to aseptic meningitis
- PLEURODENIA “devils grip” (thorax)

2.3 Echovirus
- Not point of interest for me

3. Viruses causing Diarrhea

3.1 Rotovirus
- Young children

- mainly in winter
- replicates in small intestine
- fecal-oral route of transmission

3.2 Norovirus
- older children/ adults
- leads to gastroenteritis

4. Viruses causing Exanthems (Rashes)

- humans are the only host
- highly contagious

4.1 Measles
- high infectivity
- blotchy rash behind ears
- life-long immunity after natural infection
- white KOPLIC spots in mouth

4.2 Rubella
- Pregnant women
- live attenuated vaccine
- life long immunity with natural infection

4.3 Varicella
- VZV >> chickenpox >> shingels
- latent
- 1 nerve >> localized rash

4.4 Herpes
- HSV1 >> cold sores
- HSV2 >> genital herpes

4.5 Papilloma
- warts >> hands/ feet/ genitalia
- cancer >> penis/ vagina/ cervix
- GARDASIL new vaccine

5. Viruses causing Glandular Enlargement:

5.1 Mumps
- bar/ kissing/ St. Patty's day
- bilateral inflamation of parotid glands
- meningitis, sterility, ovaritis
- spread by saliva/ respiratory secretions
- MMR vaccine >> live attenuated >> 2 shots

5.2 Epstein-Barr Virus
- herpes family
- AKA infectious mononucleosis

- mono (kissing disease) from saliva
- problems with lymph nodes
- diagnosis: atypical lymphocytes/ monospot test >> agglutinates RBCs
- confirmation >> antigen detection

5.3 Cytomegalovirus (CMV)

- affects pregnant women >> neonatal infection with jaundice, enlarged liver and spleen, mental retardation and motor disorders
- affects transplant patients >> disseminated infection can cause transplant rejection
- treatment: antivirals
- screening for CMV important during organ transplants
- CMV negative patient and CMV positive organ >> virus attacks immunocompromised patient
- CMV positive patient and CMV negative organ >> lose organ
- prophylaxis recommended

6. Hepatitis Viruses (Liver)

6.1 Hepatitis

- Which strains have and don't have vaccines? Which are blood borne, food borne etc?

Hep A

- fecal-oral route (Food boned)

Hep B

- blood borne
- average incubation period ~90 days
- infective serum 30-60 days before symptoms appear
- diagnosis >> test for Hep B surface antigen
- antibodies appear months later
- prevention includes universal precautions for blood/ body fluid handling
- proper needle handling
- Twinrix vaccine >> 3 shots

Hep C

- blood and sex
- no vaccine
- Canadian Blood Scandal

Hep D

- "Viroid"
- blood and sex
- relies on Hep B for replication
- prevention >> vaccinate against Hep B

Hep E/ Hep G

- not points of interest in my opinion

6.2 Yellow Fever Virus

- Mortality rate ~50% (relatively high)

- vector >> aedes mosquito
- prophylaxis recommended

7. Viruses affecting CNS (Brain)

7.1 Arbovirus (west nile)

- notice birds dying (crows)
- mosquitos/ stagnant water

7.2 Rabies

- fatal
- killed vaccine

8. HIV and AIDS (Immune system viruses)

- depletion of all T-helper cells
- T-helper 1 cells >> CMI
- T-helper 2 cells >> Humoral Immunity
- essentially entire immune system is depleted
- high risk for opportunistic infection leading to health problems/ death
- AIDS develops from decreasing immune status
- Prevention: screening of blood/ organ donors; testing pregnant women
- Treatment: cocktail approack >> protease inhibits maturation + transcribitase inhibits replication
- HAART >> antiviral
- bad side effects
- expensive

Food for Thought :)

Challenge: how do we vaccinate immunocompromised patients?
Possibility: Can we differentiate stem cells into T-helper 1 and T-helper 2 cells?

Lecture 10: Nosocomial (Hospital Acquired) Infections:

Definition: Infection acquired by a patient during hospitalization and having its origin in the hospital environment or in a medical procedure (includes non-preventable and preventable infections)

Chain of Infection:

- Three factors which play roles in transmission of nosocomial infections:
  1. source
  2. route of transmission
  3. host

Source:

- Environment where microorganisms multiply and disseminate from
- includes infected individuals, non-living objects, food, etc.
- endogenous >> patient's own flora
- exogenous >> outside the patient

Rout of Infection:

- The way by which micro-organisms leave the source and reach the host
- contact (direct/ person to person or indirect/ contaminated objects)
- contaminated object = FOMITE (ex: the doctor's pen light)
- water/ food/ air borne
- transmitted by a vector (ex: mosquito/ tick).

Host:

- individual infected with the micro-organism which exited the source and reached the host via one of the routes of transmission

Universal Precaution and Isolation Procedures:

- aim to prevent spread of infection to other patients/ health care providers
- universal precaution should be exercised with each and every patient, no exceptions!
    1. gloves when handling blood/ body secretions
    2. hand washing after each patient
    3. no re-capping of needles
    4. proper handling/ disposal of sharp instruments
    5. disinfecting
    6. mask and goggles >> risk of splashing

* also note importance of isolation procedures
* each hospital has an "infection control centre"

Terminology to understand:

Decontamination

- general term for removing anything harmful

Sanitization

- reducing microbial load on objects. surfaces by washing/ wiping with cleaning agents

Disinfection

- Freeing an object of microorganisms, but not necessarily of spores >> by using germicides

Ascepsis

- use of germicide on skin/ living membrane

Sterilization

- Complete elimination of microorganisms

Microorganism resistance to disinfectants:

Most resistant (least harmful) to Least resistant (most harmful)

- Bacterial spores and protazoan cysts
- Mycobacteria
- Fungi

- Vegetative Bacteria
- Enveloped viruses

*Lucky for us the most harmful microorganisms are the least resistant to disinfectants!

****Please Remember this review is meant to be used as one study tool. Also review class notes, power points and your course pack****

Good luck to everyone and feel free to email me questions at cjaco020@uottawa.ca