Infection VIVAs (Pathology)

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2014.2.C.1

Question 3 Varicella Zoster (p 353) **Subject:** Path LOA: 1	1. What are the 2 clinical conditions caused by this virus 2. Describe the pathogenesis and clinical course of infection with this virus Prompt: start with how the virus is transmitted 3. What are the complications of chicken pox	Chicken pox and shingles Starts with aerosol or direct contact spread →haematogenous dissemination → vesicular skin lesions → vesicles rupture, crust over then heal Some virus lies dormant in dorsal root ganglia and reactivated later with immunosuppression Lung → interstitial pneumonia Nervous system - encephalitis, transverse myelitis Skin and mucous membranes → shingles, bacteria superinfection Gut – necrotising visceral lesions	**Both** **Reasonable sequence** **3 to pass**

2014.1.B.2

Question 1 Measles **Subject:** Path LOA: 2	1.What organism is responsible for measles infections and how is it transmitted? 2.What type of immune response occurs in measles? 3.What are the clinical features of measles? 4. What are the complications of measles?	1.**Virus,** RNA, Paramyxo >>**respiratory transmission** 2.T cell mediated controls infection and causes rash Antibody mediated protects against reinfection 3. **fever, rash, conjunctivitis, cough/coryza,** Koplik spots, lymph nodes. 4. pneumonia, secondary bacterial infection, delayed – encephalitis, SSPE	Bold to pass Antibody mediated 3 bold to pass 2 as minimum.

2014.1.D.3

Question 3 EBV **Subject:** Path LOA: 2	(a) Describe the pathogenesis of glandular fever.	• **EBV** transmitted by close contact (saliva) • Envelope g/protein binds to B cells • Viral infection begins naso/oropharyngeal **lymphoid** tissues (esp. tonsils) • EBV accesses submucosal lymphoid tissues • B Cell infection 1) **lysis** infected cells and virion release (minority) or 2) **Latent** infection (EBV genes expressed) • Symptoms appear on initiation host immune response (cellular CD8+ cytotoxic T and NK cells) • Atypical lymphocytes (characteristic) • Reactive T cell proliferation lymphoid tissues – lymphandenopathy and splenomegaly. • IgM Ab (viral capsid Ag) and later IgG • Healthy – cease viral shedding with few resting B cells but Acquired defects may → B lymphomas	(a) To pass: EBV Lymphoid tissue Involves B (latent and lysis) and T cells
	b) What are the clinical features of glandular fever? (c) What are the outcomes of glandular fever?	(b) Classically – Fever, sore throat, lymphadenitis splenomegaly Atypical presentation common – fatigue, lymphadenopathy, hepatitis, rubella-like rash 4-6 weeks **most resolve** - some fatigue longer **Hepatic** dysfunction – j, abn. LFTs, appetite **Splenic** rupture Other systems – nervous, renal, lungs, heart. Transformation – **lymphomas**	(b) 4 clinical features to pass (c) 3 outcomes to pass

2013.1.2

Question 2 Hep B LOA: 2	1. How can Hepatitis B infection be transmitted?	1. Vertical – perinatal during childbirth Horizontal – skin or mucosal breaches - intercourse - shared needles / syringes in IVDU - blood transfusion	3/5
	2. What are the potential outcomes following ACUTE Hepatitis B infection?	**2. Recovery** >90% Fulminant hepatitis necrosis <0.5% **Chronic Hepatitis** <5% - cirrhosis 12-20% +/- hepatocellular Ca - healthy **carrier state** - non progressive chronic hepatitis <2%	**Bold to pass**
	3. What are the serum markers of ACUTE infection with Hepatitis B? Prompt: What antigens and antibodies are present during acute hepatitis B?	**3. HBeAg, HBsAg** HBV-DNA, **Anti-HBc IgM** Anti-HBe, (not Anti-HBs)	**2/3 Bold**

2012.2.2

Q3 Staph infections LOA: 1	1. Describe the virulence factors of Staph aureus. What infections do the different species of Staphylococci cause? 2. *Prompt: Name the Staphylococcal species*	a. **Surface proteins involved in adherence** – expresses receptors for fibrinogen (and others) to bind to host endothelial cells. b. **Secreted enzymes that degrade proteins** (promoting invasion and destruction) e.g. lipase degrades skin lipids associated with ability to produce abscesses **c. Secreted toxins that damage host cells** alpha toxin – membrane depolarisation/damage; beta toxin – sphingomyelinase; Exfoliative A & B toxin; Superantigens – TSS and food poisoning **S. aureus** – skin, pneumonia, osteomyelitis etc **S. epidermidis** – opportunistic eg prosthetic valves **S. saprophyticus** – UTI in women	2/3 bolded sections including toxin 2 of the 3 bolded

2012.2.4

Q3 Strep infections LOA: 1	1. What types of infections do Streptococcal bacteria cause? *Prompt: Give examples of the different strep subtypes and the infections they cause?*	1. Acute **suppurative: skin, throat, lungs and heart valves.** Group A S.pyogenes (throat, skin), Group B S.agalactiae (female genitial, neonate sepsis), α Haemolytic, S.pneumoniae (CAP), meningitis S.viridans (mouth, SABE), S.mutans (teeth)	Pus >= 2 to pass
Fri PM Q3 Strep (con'td)	2. What post infectious syndromes do streptococci cause?	2. GN, rheumatic fever, erythema nodosum	1 to pass

2012.2.4

Q4 Hepatitis C LOA: 2	1. What type of virus causes Hepatitis C? 2. What are the risk factors for acquiring Hepatitis C? 3. What is the natural course of Hepatitis C?	1. **Flaviviridae** family **RNA** virus 2. **IVDU** 54%; Multiple sex partners 36%; Recent surgery 16%; Needle stick 10%; Multiple contacts with HCV infected person10%; Health care workers 1.5% Unknown 32%; Children (perinatal) 6% (cf HBV 20%) 3. Incubation 2 – 26 weeks (mean 6 – 12); **Asymptomatic in 85%** HCV RNA detectable in 1 – 3 weeks Anti HCV Ab 50 – 70% while symptomatic Usually a mild disease **Persistent infection -> chronic hepatitis 80 – 85%** Cirrhosis 20 – 30% (5 – 20 years) Fulminant hepatitis rare	One of bold IVDU and 2 others Bolded

2011.2.1

Question 3 LOA: 1	1. What type of organisms are the Clostridia? 2. Name the organisms and the diseases they cause in humans? 3. How does botulism toxin cause disease?	**1. Gm+ve, bacilli, anaerobic, spore-forming** **2. Gas Gangrene (Perfringens), Tetanus (tetani), Botulism (botulinum), Diarrhoea (difficile)** 3.. Normally ingested. In the cytoplasm, the "A" fragment cleaves the protein "synactobrevin". Synactobrevin is needed for fusion of neurotransmitter vesicles. Results in **flaccid paralysis**	**1.** needs 3 of 4 **2.** needs 3 of 4 3. must have some idea of this plus bold

2011.2.2

Question 3 LOA: 2	1. Describe the structure of the influenza virus. 2. What are the types and subtypes Prompt:- What do H and N stand for? 3. What is the pathological basis of pandemics and epidemics?	1. **Single stranded RNA** (8 helices) Spherical capsule 2. **ABC** (determined by a nucleoprotein) **Haemagglutinin and neuraminidase** (determined by proteins on the bilipid envelope 3 **Antigenic shift** for pandemics **Antigenic drift** for epidemics Both H and N are changed by recombination of RNA from animal viruses	1. Bold to pass 2. Bold Bold to pass

2011.2.3

Question 3 LOA: 1	1. What type of bacterium is Salmonella? 2. Describe the pathogenesis of typhoid fever? 3. What are the clinical features	1. **Gram-ve bacillus** (Enterobacteriaceae family) 2. Caused by **Salmonella typhi** (endemic) and **paratyphi** (travellers). Endemic in India, Mexico, Phillipines, Pakistan, El Salvador, Haiti. Taken up by mononuclear cells in the underlying lymphoid tissue in **gut invades** M cells Reactive hyperplasia in lymph tissue. **Disseminates by blood** 3. Causes fever, anorexia, vomiting and bloody diarrhoea. BC +ve in 90% with fevers Subsequent bacteraemia with fever and flu-like symptoms	1. Bold 2. Bold 3. Reasonable response with prompting

2011.1.2

Question 3. Tuberculosis	**1. What is secondary tuberculosis?**	Pattern of disease that arises in a previously sensitised host	**previously sensitised host**
	2. How may infection occur in secondary tuberculosis?	1. May follow shortly after primary infection (<5%) **2. Reactivation of latent organisms** • Typically in areas of low disease prevalence **3. Reinfection** • Typical in regions of high prevalence	**Items 2 and 3**
	3. Describe the pathological features in the lung of secondary infection with TB.	• Locale - **apical UL in secondary** • **Area of inflammation / granuloma** / multinucleate giant cells • Central **caseous necrosis** • **cavitation** • **Healing with fibrosis and calcification** • +/- Complications include tissue destruction, **erosion of blood vessels, miliary spread, pleural effusion, empyema, fibrous pleuritis**	**Need 3 of:** • Apical site • Inflammation / granuloma • Caseous necrosis • Cavitation • Fibrosis / calcification

2011.1.3

Question 3. **Measles**	1. **Describe the pathogenesis of measles** PROMPTS: What type of virus is measles? What is the mode of transmission?	1. **Paramyxovirus (single stranded RNA)** 2. **Respiratory droplet spread** 3. Multiplies in upper respiratory tract epithelial cells 4. >lymphoid tissue where it replicates in mononuclear cells 5. haematogenous spread 6. Preventable by vaccination as only single strain. 7. Epidemics amongst un-vaccinated individuals	• **Virus** • **Respiratory droplet spread** • **+ 1 other**
	2. What type of immune responses occur in measles?	• **T cell mediated immunity** controls infection + causes rash • **Antibody mediated** protects against re-infection • epidemics in unvaccinated hosts	• cell mediated • antibody mediated
	3. **Describe some of the systemic features of measles virus infection.** Prompt: What are some complications of measles infection?	• **Rash**–blotchy, red/brown. Skin hypersensitivity reaction • Oral mucosal ulceration – **Koplik's spots** • **Croup** • **Interstitial pneumonia** • **Conjunctivitis, Keratitis**, with scarring and visual loss • **Encephalitis**; - plus SSPE, measles inclusion-body encephalitis • **Diarrhoea** with protein losing enteropathy • **Immunosuppression** • **Secondary bacterial infection**	• Rash • + 3 others

2010.2.1

Question 1.3 Hepatitis C Infection	1. What causes Hepatitis C infection? 2. Describe the clinical course of Hepatitis C infection 3. What are the risk factors for acquiring Hepatitis C?	**1.1. Flaviviridae family RNA Virus** 2.1 Incubation period 2-26 wks (mean 6-12 wks) 2.2 Acute infection usually mild or asymptomatic (1-3 weeks) 2.3 Persistent and Chronic hepatitis with exacerbations in 80% 2.4 Cirrhosis in 20-30% 2.5 Fulminant hepatic failure rare **3.1 IVDU (54%)** **3.2 Multiple sex partners (36%)** 3.3 Needle stick (10%) (risk of HCV is 1.8% v 0.3% for HIV) 3.4 HCW (1.5%) 3.5 Blood Transfusion (in the 1980's), 3.6 Vertical, 3.7 Unknown (32%)	1. Bold 2. 3/5 3. 3/7
	Additional question for good candidates. After completion of 5 questions 4. What features of the Hepatitis C virus make vaccine development difficult?	4.1 Highly stable core, **extremely variable envelope (E protein)** 4.2 RNA polymerase inherently unstable; frequent mutations, multiple *quasispecies* found in any one pt 4.3 Genomic and Antigenic variability 4.4 Actively inhibits interferon mediated cellular response at many levels	4. 2/4

2010.2.2

Question 2.4 Cholera	1. What is the causative organism of cholera? 2. Describe the pathogenesis of cholera (Describe how the enterotoxin causes diarrhoea).	1. **Vibrio cholera = gram neg bacteria** (comma shaped/flagellate) 2. Pathogenesis 2.1. **Non invasive** 2.2. **Flagella proteins** for attachment & colonization **2.3. Preformed enterotoxin** 2.3.1. Cholera enterotoxin • 5 B subunits • 1 A subunit 2.3.2. B subunit **binds** to intestinal (mainly duodenum/jejunum) – epithelial cells • Retrograde transport in ER 2.3.3. A subunit Tx to cytoplasm • A subunit activates G protein • Stimulates adenyl cyclase→ c-amp • Opens cystic fibrosis transmembrane conductance regulator (CFTR) • **Releases Cl^- into lumen** o **secretion of HCO_3, Na and H_20** o **massive diarrhoea which overwhelms colonic resorption**	1. Bold 2. Need 4 bold to pass

2010.2.4

Question 4.5 E. coli Gastroenteritis	List the types of E. Coli enteritis and describe their features	**1.1 Enterotoxic E coli** (ETEC) **1.1.1** Food and water, traveller's **1.1.2** **LT heat labile toxin**, adenyl cyclise **-> inc cAMP -> inc Cl- secretion** and **decr absorption (cholera like)** **1.1.3** ST **heat stable toxin**, guanylate cyclase **-> incr cGMP** **1.2 Enterohaemorrhagic E coli** (EHEC) **1.2.1** **Beef esp. ground**, milk vegetable **1.2.2** **O157:H7** and non O157:H7 **1.2.3** **Shigella like toxin** **1.2.4** Large outbreaks, **bloody diarrhoea, haemolytic uraemic syndrome** **1.2.5** Thrombotic Thrombocytopenic purpure (**2%**) **1.3 Enteroinvasive E. Coli** (EIEC) **1.3.1** Food, water, person to person **1.3.2** No toxins, invades mucosa, colitis **1.4 Enteroaggregative E. coli** (EAEC) **1.4.1** Adheres via **adherence fimbriae.** **1.4.2** Dispersin (removes –ve charge/ protection) **1.4.3** Shigella like toxin and ETEC ST toxin **1.4.4** Non bloody diarrhoea, prolonged in AIDS	2 of 4 groups to pass 1 feature of any two

2010.1.2

Question 5: Influenza	i) Describe the structure and classification of influenza viruses	**ssRNA, bound by nucleoprotein that determines type (A, B or C) and a lipid bilayer that contains both haemagglutinin and neuraminidase** (determining subtype eg H1N1)	Need RNA and major types
	ii) What is the difference between antigenic drift and shift?	Only in influenza type A **Drift – mutation** of the haemaglutinin and neuraminidase antigens allowing escape from most host antibodies (epidemic) **Shift – antigens replaced via recombination of RNA** segments with those of animal viruses (pandemic) Types B and C do not show drift or shift, mostly infect children, who develop antibodies preventing re-infection	Bold
	iii) How does the human body clear a primary influenza virus infection?	**2 mechanisms – cytotoxic T cells and macrophages** cytotoxic T cells kill virus infected cells, an intracellular antiinfluenza protein (Mx1)is induced in macrophages by cytokines IFN-a and IFN-b. Future infection is prevented (haemagglutinin Ab) and ameliorated (neuraminidase Ab)	Bold to pass

2010.1.3

Question 4: Hepatitis D	i) Describe how the Hepatitis D virus infects the human body	RNA virus **Must always be in conjunction with Hep B** 1) **acute infection –** indistinguishable from classical acute Hep B.) Exposure to serum containing both Hep B and D. HBV must establish first to provide HBsAg necessary for development of complete HDV viridons 2) **superinfection.** -chronic HBV carrier exposed to new inoculum of HDV. Disease develops 30-40 days later 3) helper-independent latent infection- in liver transplantation patients	Bold to pass
	ii) Prompt: *Superinfection is one of the ways that Hepatitis D can infect the human host.* How does superinfection with HDV manifest?	1) severe acute hepatitis in previously unrecognised HBV carrier 2) exacerbation of preexisting mild chronic hepatitis B 3) 80-90% chronic progressive disease and cirrhosis	Need one
	iii) How is Hepatitis D infection diagnosed?	**IgM anti-HDV** – most reliable marker of recent HDV exposure but late and short lived HBV an HDV coinfection – best with IgM against both HDAg and HBcAg *2 phases –* *acute phase – active HDV replication, suppression of HBV, high ALT levels* *chronic phase – HDV replication decreases, HBV replication increases, ALT levels fluctuate, progression to cirrhosis and hepatocellular cancer* *HDV RNA detectable in blood and liver just prior and in early days of acute symptomatic disease* *In chronic delta hepatitis, HBsAg is present and IgM and IgG anti-HDV antibodies persist for months*	At least one

2009.2

Qn 1	What are streptococci?	**Gram-positive cocci** growing in pairs or chains. Facultative or obligate anaerobes. Cause variety of suppurative infections and immunologically mediated post-streptococcal syndromes.	Bold to pass
Qn 2	Name some of the different types of streptococci and give examples of diseases they cause.	Alpha haemolytic - **S. pneumonia - pneumonia** - meningitis S viridans - endocarditis β Haemolytic - **Group A. (Pyogenes)** pharyngitis - scarlet fever - erysipelas - Impetigo - Rheumatic fever - Toxic Shock Syndrome - Glomerulonephritis Group B. (Agalactiae) - neonatal sepsis and meningitis - chorioamnionitis Strept. mutans - dental caries	3 major type/group + 6 diseases to pass
Qn 3	What factors in streptococci contribute to their virulence? .	Capsules pyogenes, pneumoniae M Protein prevents phagocytosis (anti M protein AL → Rh.F.) Complement C5a peptidase Pneumolysin lyses target cells (S pneumoniae) activates complement Pyrogenic exotoxin- rash and fever High MW glucans plaque formation aggregation of platelets Sucrose → lactic acid (S. mutans).	Any 3 to pass Capsule important.

2009.1

Question 3:	**What are the microbiological features of *Neisseria*?**	1. Aerobic 2. Gram negative diplococci 3. Coffee bean shaped 4. Require chocolate blood agar 5. 13 serotypes of *N. meningitidis*	Prompt: What are the staining characteristics of *Neisseria?*

2009.1

Question 4: **Influenza**	**How does the Influenza virus cause pneumonia?**	1. **Attachment of virus to upper resp tract epithelium** 2. Necrosis of cells followed by inflammatory response 3. Interstitial inflammation with outpouring of fluid into alveoli 4. **Secondary infection by Staph / Strep**	Need bolded
	How does Influenza A cause epidemics and pandemics	Mutations of Influenza A haemagglutinin and neuraminidase allow virus to escape host antibodies (**antigenic drift**) and epidemics, whereas replacement of these with animal-derived RNA segments leads to new virus (**antigenic shift**) and pandemics	Needs bolded

2009.1

Question 3: **Hepatitis C**	**What are the routes of transmission of Hepatitis C virus?**	1. **Blood inoculation with IV drug use** 60%. 2. Unknown 3. Transmission via blood products pre1991 10% 4. Dialysis < 5% 5. Occupational exposure < 5% 6. Sexual transmission infrequent 7. Vertical transmission very low.	Need bold and 2 other?
	What are the potential outcomes of infection?	1. Acute Infection generally asymptomatic, rarely fulminant hepatitis 2. **85% progress to chronic persistent hepatitis** 3. 15% resolve completely 4. **20% of chronic infection progress to cirrhosis** 5. Some cirrhotics develop **Hepatocellular carcinoma**	Need bold

2009.1

Question 3: **Neisserial infections**	**What are the two clinically significant *Neisseria*?** **Describe the pathogenesis of a *N. meningitidis* infection**	1. *meningitidis* 2. *gonorrhoeae* 1. Respiratory spread 2. Common coloniser of the oropharynx 3. (10% of the population at any one time) 4. Colonisation lasts for months 5. Immune response leads to protection against that strain 6. Invasive disease crosses respiratory epithelium to enter blood 7. Capsule of *Neisseria* reduces opsonisation & protects against destruction by complement proteins 8. Outbreaks in young people living in crowded quarters who encounter new strains	Both Need 5/8 Prompt: How does it spread?

2008.2

3. Tuberculosis	Describe the **pathogenesis** of tuberculosis in a previously **unexposed immunocompetent** person Prompt if doesn't mention airborne.	**Infection by M. tuberculosis airborne** • M. tuberculosis usually person to person airborne droplet spread **M tuberculosis enters alveolar macrophages and replicates** • Enters alveolar macrophages and replicates by blocking phagosome/lysosome fusion leading to bacteraemia (person generally asymptomatic or mild flu like illness **Immunity through T cell mediated delayed type hypersensitivity reaction that also causes hypersensitivity and tissue destruction- in particular granuloma formation and caseation** • About 3 weeks later T cell activation via MHC antigens on macrophages and IL-2 leading to macrophage becoming bactericidal (thru IFN-γ) This macrophage response also causes tuberculin positivity and formation of granuloma and caseation by recruiting monocytes ("epithelioid histiocytes") **Re- exposure or reactivation causes heightened immune reaction as well as tissue destruction** • Infection may be contained or may progress and may reactivate with immunosuppression from any cause	Highlighted

2008.2

3. Malaria	1. What micro-organisms cause malaria?	Parasitic protozoa **Plasmodium falciparum**, vivax, ovale, malarie	Falciparum +1
	2. How does Plasmodium falciparum infection differ from other forms of malaria? Prompts: How does it compare clinically? By what mechanism?	All do: sporozoite→liver→merozoites formed → release & bind to RBC→ Hb hydrolysed → trophozoite→ schizont → merozoite/gametocyte *P. falciparum:* **infects RBCs of any age**, causing **clumping/rosetting** so ischemia, high cytokine production, **high level parasitemia**, severe anemia, cerebral symps, renal failure, pul oedema, death *Others*: infect only new or old RBCs, P vivax & ovale form latent hynpnozoites (relapses), low parasitemia, mild anemia, rarely splenic rupture, nephrotic synd	2/3 Highlighted and 1 clinical feature
	3. What factors can make people less susceptible to malaria?	**Inherited alterations in RBCs: HbS** trait, HbC, Duffy Ag neg Repeated exposure stimulates immune response: Ab and T lymphocytes (P falc avoids this), HLAB53	Highlighted

2008.2

3. Candidiasis	1. What is the clinical spectrum of candida infection?	(Benign commensal) **Superficial mucosal infn –** mouth, vagina, oesophagus **Superficial cutaneous infn –** intertrigo, nappy rash, balanitis, folliculitis, paronychia, onychomycosis Chronic mucocutaneous (T-cell defects, endocrinopathy) **Invasive (disseminated) –** myocardial/ abscess/endocarditis, cerebral abscess/meningitis, renal/hepatic abscess, endophthalmitis, pneumonia	Highlighted – something from each category
	2. What mechanisms enable candida to cause disease? Prompt: What are the virulence factors?	1) **Phenotypic switching** to adapt rapidly to changes in host environment 2)**Adhesion to host cells -** imp. determ. of virulence –via adhesins (several types) 3) **Production of enzymes** (aspartyl proteases and catalases) degrade extracellular matrix proteins and may aid intracellular survival 4) secretion of adenosine – blocks neutrophil degranulation	1/3 Highlighted

2008.1

Q3. Hepatitis A	Describe the clinical course of Hepatitis A infection.	1) Oral faecal transmission. 2) Incubation period: 2-6 weeks. 3) No carrier state or chronic hep or cause hepatocellular Ca. 4) Rarely causes fulminant hepatitis, and so the fatality rate is about 0.1%.	Pass criteria: provide 3/4 Prompt: mode of transmission.
	How do the serological markers change with time in Hep A infection?	1) IgM anti HAV appears at the onset of symptoms. 2)Faecal shedding of the virus ends as IgM titre rises (2-12 weeks). 3) IgM Ab (months) 3) Replace by IgG anti HAV (years) . Encourage graph	Encourage graph.

2008.1

Q3. Hepatitis C	Describe the potential outcomes of acute hepatitis C infections in adults.	1)Acute fulminant rare 2)15% resolve 3)85% chronic - >80% stable/20% cirrhosis (50% mortality) hepatocellular Ca	3 major points with most > chronic fulminant) and potential for cirrhosis/Ca
	How does the serology for Hepatitis C infection change in case of resolution?	1)Incubation period (2-26 weeks) 2)HCV-RNA (detectable for 1-3 weeks co-incident with transaminitis) 3)Anti HCV antibodies emerge. Only about 50% detectable during symptomatic acute infection. Remainder after 3-6 weeks. IgG/IgM. IgG persists.	All major points & 'window' when both virus & Ab may be – ve. Diagram encouraged.

2008.1

Q3. Clostridial infections	Name some clostridial diseases and causative organisms.	1) Tetanus (lockjaw) – Clostridium tetani 2) Botulism (paralytic food poisoning) – Clostridium botulinum 3) Gas gangrene, necrotizing cellulitis – Clostridium perfringens, C. septicum 4) Pseudomembranous colitis – Clostridium difficile	Pass: Require 2 out of 4
	What is the pathogenesis of gas gangrene (C. perfringens, C. septicum)	Release enzymes – hyaluronidase; collagenase Virulence factors – TOXINS <u>α-toxin</u> - multiple actions - phospholipase C: degrades membranes; muscle; RBC - release phospholipid derivatives: ITP; prostaglandins - these cause derangement in cell metabolism and cell death	At least 2 & α-toxin