ACEM PRIMARY 2010/2 PATHOLOGY VIVA Thursday (September 16) Session: Morning *Candidate Number*…… AGREED MARK……

TOPIC	QUESTION	ESSENTIAL (BOLD) KNOWLEDGE (UNBOLD)	NOTES
Question 1.1 Vascular Changes of Inflammation	1. Describe the vascular changes in acute inflammation 2. What are the mechanisms of increased vascular permeability?	1.1. **Vasodilatation:** opening of arterioles and capillary beds mediated by histamine and Nitric Oxide leading to increased blood flow 1.2. **Increased vascular permeability** 1.3. **Stasis:** due to PP permeability and increased viscosity 2. 2.1. Endothelial contraction / retraction: gaps in venules due to histamine and leukotrienes < 30mins, immediate transient response eg.ultraviolet radiation and kinins and leukotrienes 2-12hrs, delayed prolonged leakage eg. late appearing sunburn 2.2. Direct vascular endothelial injury eg. in severe burns, microbial toxin injury, amplified by neutrophil activation, rapid onset but may last days 2.3. Leukocyte mediated leakage, in venules and pulm capillaries, long lasting for hours 2.4. Trancytosis increased Tx of fluid and protein thru endothelial cell, VEGF	1. All 3 2. 2 out of 4
Question 1.2 Antibody Mediated Hypersensitivity	1. What is antibody - mediated hypersensitivity? 2. Describe the mechanisms which mediate the hypersensitivity response 3. List an example or examples for each mechanism	1. **Caused by antibodies that react with antigens present on cell surfaces or in the extracellular matrix** Antigens can be intrinsic to the membrane or matrix or extrinsic eg. Drug metabolite 2. Mechanism of hypersensitivity response 2.1. **Opsonisation and phagocytosis: IgG antibodies opsonise cells plus complement activation** generates C3b and C4b recognized by phagocyte Fc and protein receptors resulting in **phagocytosis and destruction of opsonised cells** **Examples:** transfusion reaction, erythroblastosis fetalis, autoimmune haemolytic anaemia, agranulocytosis, thrombocytopaenia, drug reactions when a drug acts as a hapten 2.2. **Complement and Fc receptor mediated inflammation: antibodies bind to fixed tissue** such as basement membranes, extracellular matrix … **activates complement** … generate by-products particularly chemotactic agent C5a … direct PMN migration and C3a and C5a **= increase vascular permeability. PMNs activated** by C3a and Fc receptors… release of pro- inflammatory substances like prostaglandins, production of lysosomal enzymes, reactive O2 species **Examples:** glomerulonephritis, vascular rejection in organ grafts, vasculitis caused by ANCA, Goodpastures 2.3. **Antibody mediated cellular dysfunction:** antibodies directed against cell surface **receptors impair or dysregulate function without causing cell injury or inflammation** **Examples:** myasthenia gravis, Graves's disease, insulin resistant diabetes, pemphigus vulgaris **2.4. Antibody dependant cellular cytotoxicity** **Examples:** IgG coats cells, effector cells such as monocytes, neutrophils, eosinophils and NK cells then bind and lyse cells without phagocytosis, role in specific diseases uncertain.	1. Bold 2. Bold 2/4 3. 2/4
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,	1. Bold 2. 3/5 3. 3/7

		3.7 Unknown (32%)	
	Additional question for good candidates. After completion of 5 questions		4. 2/4
	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	
Question 1.4 Disseminated Intravascular Coagulation	1. Describe the pathophysiology of "disseminated intravascular coagulation"? ("Trigger" can be a prompt)	1. 2 major mechanisms **trigger** DIC: 1.1 release of tissue factor into circulation 1.2 widespread injury to the endothelial cells 1.3 Acute, subacute or chronic thrombo-haemorrhagic disorder characterized by 1.3.1 excessive activation of coagulation leading to 1.3.2 formation of **thrombi in the microvascular** circulation 1.3 .3 secondary activation of **fibrinolysis causing bleeding** 1.3.**4** **consumption of platelet**s, fibrin and coagulation factors	1. 1 trigger and 2/3 bolds
	2. What are some of the important causes and triggers of severe DIC?	2.1 **Obstetric complications** (eg amniotic fluid embolism, FDIU) responsible for approx 50% cases 2.2 Malignant neoplasms (33% cases) **2.3 Sepsis** **2.4 Major trauma**, severe burns, extensive surgery 2.5 Transfusion reaction 2.6 Most mild cases probably due to sepsis, esp in elderly, but not usually diagnosed – low plts	3. 3/6
Question 1.5 Post Streptococcal GN	1. Describe the aetiology and pathogenesis of post streptococcal glomerulonephritis.	1.1 G**roup A β-hemolytic streptococci** (eg: 90% types 12, 4, and 1) 1.2 Typically post pharyngeal/skin infections (impetigo) - sometimes epidemic, partic in overcrowded insanitary conditions 1.3 An **immunologically mediated disease** ? Type 2/ or 3 type e.g. ? Circulating or antigen deposit disease. 1.4 Granular **immune deposits in the glomeruli** (IgG & C3) - partic GBM- leading to leaking glomeruli. 1.5 Streptococcal antigen found in the glomeruli. 1.6 Complement activation – low serum complement **1.7** Elevated titres of anti streptococcal Ab 1.8 Nephritis associated streptococcal plasmin receptor NAPlr, Strep pyogenic exotoxin B (SpeB), zSPeb	1. 2 x Bold + 1 others
	2. Describe the clinical features of post Streptococcal GN.	1. 1 to 4 weeks after a streptococcal infection of the pharynx or skin (impetigo). 1.1. Malaise, fever, nausea, oliguria, and **haematuria** 1.2. Red cell casts, mild **proteinuria** (usually < 1 gm/day), periorbital and other **oedema**, mild to moderate **hypertension** 1.3. 95% will recover quickly in 1-3 weeks, 4 % chronic, 1% severe acute renal failure. Adult onset has worst prognosis 1.4. Depleted C3 and almost always Strep Ags.	2. 2 x Bold + 2 others

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TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 2.1 Hypertrophy	1. What is tissue hypertrophy? 2. What are examples of hypertrophy (Prompt: How is it classified??) 3. How is hyperplasia different form hypertrophy?	1.1. **Increase in cellular size not number** leading to overall organ/tissue size increase 1.2. Cell size increased by **more structural components and** increased synthesis of cellular proteins 1.3. Triggered by increased functional demand or stimulation by hormones or growth factors 1.4. Can be selective hypertrophy of specific sub-organelles 2. Examples 2.1. **Physiological** skeletal muscle enhancement through training or uterus under influence of hormones such as oestrogen 2.2. **Pathological** such as cardiomegaly in hypertension and CCF (has an **upper limit** after which regression occurs -> cell injury -> apoptosis/necrosis) 3. Hyperplasia involves an **increase in the number** of cells.	1. Bold 2. Bold (+ 1 example of each) 3. Bold
Question 2.2 Role of Platelets in Haemostasis	1. What are the 2 main roles of platelets in haemostasis? 2. How is the primary haemostatic plug formed?	**1.1. Primary Haemostatic Plug** **1.2.** Provides surface to **recruit and concentrate activated coagulation factors** **2.** After vascular injury, platelets contact exposed ECM eg. collagen, adhesive glycoprotein, vWF 2.1. **Adhesion** – via glycoprotein 1b (GpIb) receptor to vWF forming bridge between plat and ECM collagen 2.1.1. necessary to overcome high shear force of blood flow, deficient in vW disease or Bernard-Soulier syndrome 2.2. Activation resulting in shape change and s**ecretion** – **granule release** (**ADP**, **TxA2**). 2.3. **Aggregation** – ADP potent activator of platelet aggregation and +ve feedback for more ADP release. Agonist binding causes intracellular protein phosphorylation cascade => degranulation, including dense body content release of **Ca^{++}**, required for coagulation cascade. Platelet activation causes appearance of negatively charged phospholipids on surface => bind Ca, critical nucleation sites for assembly of coagulation factor complexes. 2.4. TxA2 amplifies platelet aggregation **=>** leads to formation of primary haemostatic plug. 2.5. Aggregation reversible at this stage but not after next stage of stabilization via coagulation cascade with formation of thrombin.	1. **Bold** to pass 2. **4/7 Bold** to pass
Question 2.3 Ulcerative Colitis	**1.** What are the pathological features of Ulcerative Colitis? **2.** What extra-intestinal manifestations occur in ulcerative colitis?	**1.1.** One of two disorders that compromise **inflammatory bowel disease (IBD)** **1.2.** Severe ulcerating inflammatory disease **1.3.** Limited to **colon and rectum.** 1.3.1. Continuous distribution (Starts in colon and extends continuously – No skip lesions) 1.3.2. Extends only into mucosa and submucosa (ie not trans mural) 1.3.3. Pancolitis if entire colon affected, limited distal disease eg ulcerative proctitis 1.4. Superficial broad based ulcers 1.5. Pseudopolyps 1.6. Malignant potential 1.7. Toxic megacolon 2. Extra-intestinal Manifestations 2.1. Polyarthritis, 2.2. sacroiliitis, ankylosing spondylitis 2.3. Uveitis 2.4. Skin lesions 2.5. Pericholangitis 2.6. Primary sclerosing cholangitis	**1.** Bold (+ 2) 2. 4 for pass

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 HCO3, Na and H_20** o **massive diarrhoea which overwhelms colonic resorption**	1. Bold 2. Need 4 bold to pass
Question 2.5 Parkinsonism	1. Describe the clinical features of Parkinsonism. (Prompt: How do Parkinsonian patients look?) 2. What are the causes of Parkinsonism? (Prompt: what part of the brain is affected?) 3. Outline the possible pathogenesis of Parkinson's Disease.	1. Diminished facial expression, stooped posture, slowness of voluntary movement, festinating gait (progressively shortened, accelerated steps), rigidity and a "pill-rolling" tremor. **2. Conditions that cause damage to the <u>nigrostriatal dopaminergic system</u>** 2.1. Parkinson disease 2.2. Post-encephalitic 2.3. Familial forms (rare – auto dominant & recessive) 2.4. trauma/ injuries 2.5. Drugs – dopamine antagonists/toxins/pesticides 2.6. Multiple system atrophy, progressive supranuclear palsy 3. Possible pathogenesis – no unifying pathogenic mechanism identified 3.1. Misfolded protein/stress response triggered by α-synuclein aggregation 3.2. Defective proteosomal function due to the loss of the E3 ubiquitin ligase parkin 3.3. Altered mitochondrial function caused by the loss of DJ-1 and PINK1 3.4. Genetic variants with gene defects 3.5. Possible damage to dopaminergic cells from toxins drugs/AI conditions	1. 3 of 6 2. Bold + 2

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TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 3.1 Type 1 (Immediate) Hypersensitivity	1. What are the features of Type 1 hypersensitivity? 2. What are the actions of mast cell mediators in Type I Hyeprsensitivity (and give examples) 3. What is the late phase reaction	**1.1. Immediate reaction, , previously sensitised individuals, IgE mediated** 1.2. **Mast cell** and or basophils involved 1.3. **Mediators** involved include Histamine, other amines, enzymes proteases, proteoglycans, heparin, leukotrienes, C4, PAF, Prostaglandins, Cytokines **2.1 Cellular infiltration** – leukotrienes, chemotaxis, PAF, Cytokines 2.2 **Vasoactive effects –** Hist, PAF, Leukotrienes, PG D4 **2.3 Smooth muscle spasm –** leukotrienes, histamine, PG, PAF **3 Ongoing inflammatory reaction without additional exposure to triggering ag**	1. 3/5 bold 2. Histamine + 2 others + reasonable actions 3. Ongoing
Question 3.2 Angiogenesis	1. What is angiogenesis? 2. Please give some examples? 3. What steps are involved in angiogenesis from pre existing vessels?	1. **The process of blood vessel formation in the adult.** 2 methods 1.1. Branching and extension of existing vessels 1.2. Recruitment of endothelial progenitor cells (EPCs) 2. Wound healing, chronic inflammation, proliferating endometrium, tumours, etc 3. Steps in angiogenesis 3.1. Vasodilation 3.2. Proteolytic degradation of basement membrane 3.3. Endothelial cells migrate to angiogenic stimuli 3.4. Maturation 3.5. Capillary formation 3.6. Recruitment of periendothelial cells for support structure formation **4.** Inhibitors such as endostatin are released by proteinases (This is a small fragment of collagen that inhibits endothelial proliferation and also angiogenesis)	1. **Bold** and one other 2. **Any 2** 3. **Any 3**

Question 3.3 Pseudo-membranous Colitis	1. Describe the pathogenesis of pseudomembranous colitis. 2. What are the clinical features of pseudomembranous colitis? 3. What is the pseudomembrane?	**1.1. Disruption of normal bowel flora (ab's – esp. 3rd gen ceph) allowing overgrowth of *C. difficile*** **1.2. *C. difficile* elaborates toxins that cause:** 1.2.1. Ribosylation of small GTPases 1.2.2. Disruption of epithelial cytoskeleton 1.2.3. Tight junction barrier loss 1.2.4. Cytokine release 1.2.5. Apoptosis 1.3. Denuded surface epithelium 1.4. Superficial lamina propria contains dense infiltrate of neutrophils & occasional fibrin thrombi in capillaries 1.5. Damaged crypts are distended by mucopurulent exudates that erupt "volcanically" 1.6. Coalesce to form the pseudomembrane 2. Causes fever, (leukocytosis), profuse watery diarrhoea, abdo pain **3. Pseudomembrane is an adherent layer of inflammatory cells and debris at sites of colonic mucosal injury**	1. Toxin + one otherbold + 1 other (1.3 to 1.7) 2. 2/3 3. bold
Question 3.4 Cholecystitis	1. Describe the pathogenesis of acute calculous cholecystitis 2. How does acalculous cholecystitis differ from this? 3. Describe the clinical features of acute cholecystitis.	1. Acute Calculous (90% of all) 1.1. **Obstruction by stones,** stasis- activates hydrolases 1.2. Lecithins -> (mucosal Phospholipases) **-> lysolecithins** 1.3. Disrupts glycoprotein mucous -> **epithelium exposed to bile salts** 1.4. Prostaglandin release -> **inflammation,** mucosal and mural 1.5. **Dysmotility** & **raised intraluminal pressure** **1.6. Bacterial infection secondary to stasis** **2. Acalculous (10%) – rarer, in predisposed individuals, slower often masked** **2.1. Ischaemia, end arteries (cystic)** **2.2. Other promoting features – sludging micro-crystals, stasis, local inflammation, distension** 2.3. Sepsis with hypotension, immunosuppression, major trauma and burns, diabetes, infection, severe atherosclerosis (drugs/ABs- ? vasculitic). 3. Right upper quadrant or epigastric pain, 3.1. Mild fever, anorexia, tachycardia, sweating, nausea, and vomiting, tender RUQ (Murphy's)	1. 3/6 2. 3/6 3. 4/7
Question 3.5 Osteoarthritis	1. What factors lead to osteoarthritis 2. Describe the pathological changes that occur in an affected joint 3. Describe the major clinical features of osteoarthritis	1.1. Genetic & environmental (mechanical) 1.2. Age – virtually ubiquitous (80-90%) after 65 1.3. Other exacerbating diseases e.g. Obesity, diabetes, injury, abnormal joints, 2.**Chondrocyte injury** 1.3.1. Early OA: **chondrocytes proliferate (cloning) and secrete inflammatory mediator**s, collagens, proteoglycans, and proteases which initiates secondary inflammatory changes. 1.3.2. Later OA: repetitive injury and chronic inflammation lead to chondrocyte drop out, marked loss of cartilage, and extensive **subchondral bone changes** 3.Mostly **asymptomatic** <50y.o. 1.4. Deep, achy **pain** worse with use, **morning stiffness**, **crepitus,** and **limited ROM** 1.5. **Oligoarthritis 95%** (occas generalized/early) 1.6. Impingement on spinal foramina by osteophytes results in cervical and lumbar nerve root compression and **radicular pain**, muscle spasms, **muscle atrophy**, and **neurologic deficits**. 1.7. Common: hips, knees, lower lumbar and cervical vertebrae, PIP, DIP of the fingers, 1st carpoMC joints, and 1st TarsoMT joints. Not wrists, elbows, shoulders	1. 2/4 answers 2. 2/3 bold, 3. 2/4

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TOPIC	QUESTION	ESSENTIAL KNOWLEDGE	NOTES
Question 4.1 Cellular Events of Inflammation	1. How do leucocytes get to an area of acute inflammation? 2. What is the role of leukocytes in acute inflammation?	1.1 **Margination** of WCC in vessels, rolling and adhesion to endothelium (pavementing) (Selectins) 1.2 **Migration** and **diapedesis** across endothelium (PECAM1, CD31, Integrins) 1.3 **Migration towards chemotactic** stimulus in tissue (bacterial products, cytokines, IL8, C5A) 2 2.1 **Recognition and attachment** to materials (opsonins) mediated by receptors 2.2 Killing of microbes: **phagocytosis /engulfment /killing and degradation** (H2O2-MPO-Halide) 2.3 **Release of products** – Amplify the inflammatory reaction (lysosomal enzymes, reactive oxygen/nitrogen)	1. All Bold 2. 3/5 Bold
Question 4.2 Acute Pancreatitis	1. What is the aetiology of acute pancreatitis? 2. What is the suggested pathogenesis of acute pancreatitis? 3. What are the laboratory findings of acute pancreatitis?	1.1 Metabolic – **Alcohol** 5% (UK), 65% (US), M:F = 6:1, drugs eg. azothioprine, hyperlipoproteinemia, hypercalcaemia, 1.2 Genetic – trypsinogen and trypsin genes 1.3 Mechanical – **Gallstones** 35-60%, M:F = 1:3, trauma, iatrogenic/intraoperative/ERCP 1.4 Vascular – shock, atherosclerosis, vasculitis 1.5 Infectious – mumps 2. 2.1 **Autodigestion of pancreatic substance by inappropriately activated pancreatic enzymes** 2.2 3 mechanisms 2.2.1 **Pancreatic duct obstruction** eg. by impacted gallstone => accumulation of lipase in interstitium => local fat necrosis => release of proinflammatory cytokines => leaky vessels + oedema => vascular insufficiency and ischaemic damage to acinar cells 2.2.2 **Primary acinar cell injury** eg. alcohol, mumps, trauma, drugs, organ insufficiency aftershock/ischaemia 2.2.3 **Defective intracellular transport of proenzymes within acinar cells** – digestive enzymes and lysosomal hydrolases intermingled causing release of activated enzymes. Human mechanism not clear. 3 **3.1 Marked elevation of serum amylase** in first 24 hours **3.2 Rising serum lipase** within 72-96 hours **3.3** Glycosuria – 10% cases **3.4** Hypocalcaemia – poor prognostic sign if persistent **3.5** Leukocytosis **3.6** Acute renal failure	1. **Bold** + 2 of the other causes from different groups **2.1** **Bold** to pass **2.2** **2 of 3** bold **3.** **Bold** + 2 others to pass
Question 4.3 Abdominal Aortic Aneurysm	1. Describe the pathogenesis of an aneurysm 2. What are the clinical consequences of an AAA? 3. What is the risk of rupture of an AAA?	1. **Structure or function of the vascular wall connective tissue is compromised** 1.1. **Poor intrinsic quality of the vascular wall connective tissue eg Marfan syndrome**, Ehlers-Danlos 1.2. **Collagen degradation vs synthesis by local inflammation (** proteolytic enzymes**) eg atherosclerotic plaque, vasculitis,** 1.3. **Loss of vascular smooth muscle cells** or the inappropriate synthesis of noncollagenous or nonelastic ECM (cystic medial degeneration) 2. 2.1. **Rupture** into the peritoneal cavity or retroperitoneal tissues with massive, potentially fatal haemorrhage 2.2. **Obstruction of a branch vessel** resulting in ischemic injury, eg. iliac, renal, mesenteric, or vertebral arteries 2.3. **Embolism** from atheroma or mural thrombus 2.4. **Impingement** on an adjacent structure, e.g. ureter, vertebrae 2.5. Nothing (if < 4cm and no embolic complic's) 3. Related to size - 3.1 4 cm or less in diameter nil 3.2 between 4 and 5 cm 1% per year 3.3 between 5 and 6 cm 11% per year 3.4 greater than 6 cm in diameter 25% per year	1. 2/3 **bold**, 2 examples 2. 3 out of 5 3. Low < 5cm, much higher > 5cm

Question 4.4 Iron Deficiency Anaemia	**1.** What is the aetiology of Fe deficiency anaemia?	**1.1. Chronic blood loss – GIT, menorrhagia** 1.2. Increased requirement – pregnancy 1.3. Dietary deficiency – vegetarians 1.4. Impaired absorption – celiac	1. Bold + 1
	2. What are the laboratory findings in Fe deficiency anaemia?	2. 2.1. **Microcytic hypochromic anaemia** (low Hb) 2.2. Low S. Fe levels 2.3. Low S. Ferritin levels (correlates well with body iron stores) 2.4. High TIBC (high transferrin levels) 2.5. Low Transferrin saturation levels	2. Bold +3
	3. What are the clinical features of Fe deficiency anaemia?	3. 3.1. **General** - pallor, weakness, lethargy, fatigue, SOBOE, angina 3.2. Features of blood loss – GI, menorrhagia 3.3. **Specific** features – koilonychia, alopecia, glossitis, pica	3. At least 5 from 2 groups
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