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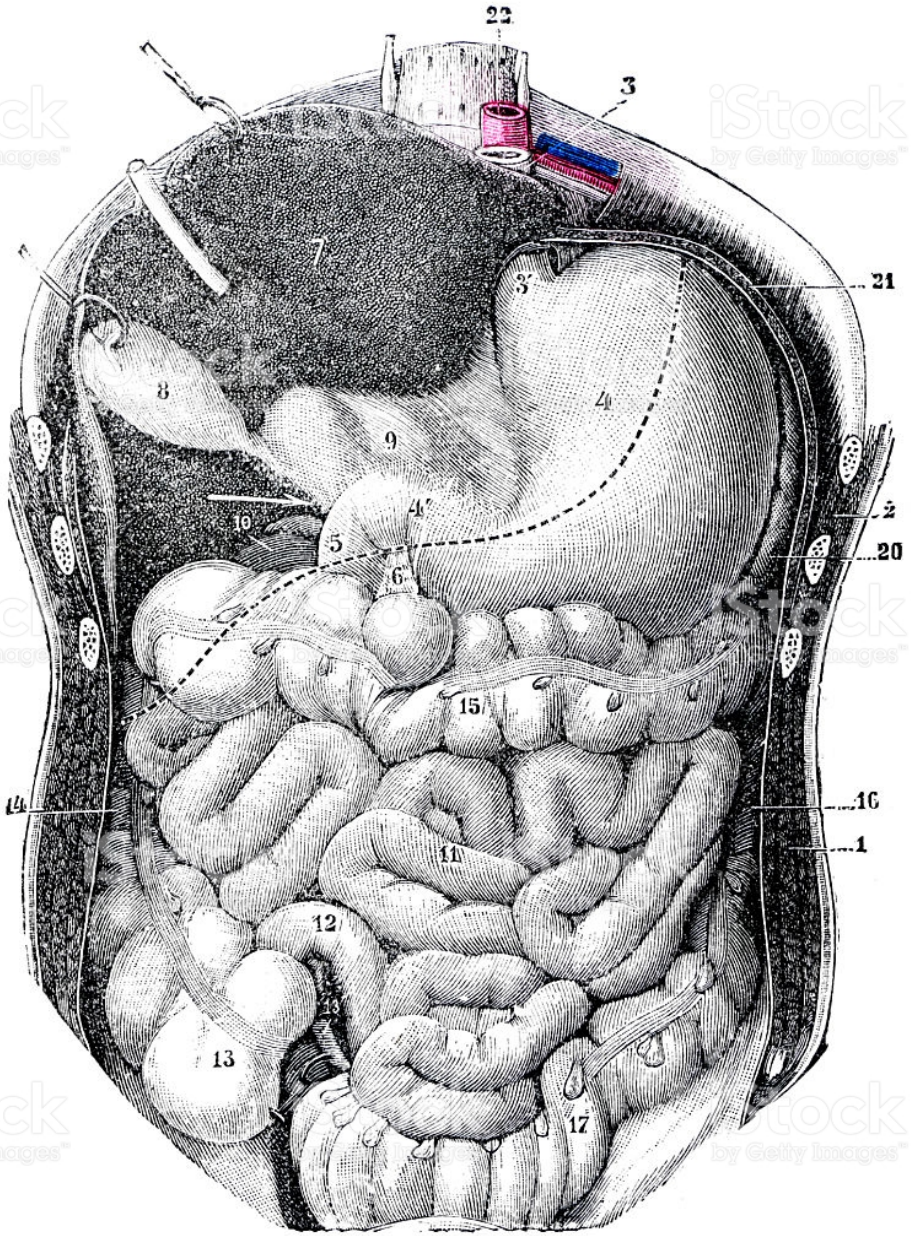


Bilirubin 2017-2-D

Stem: Moving on to Physiology			
Question 2 Bilirubin **Subject:** Physiology LOA: 1	(a) How is bilirubin produced in the body?	By breakdown of haemoglobin (heme is converted to biliverdin and then on to bilirubin)	Concept to pass
	(b) How is bilirubin metabolised?	• Bound to albumin in the circulation • Dissociates in the liver and free bilirubin enters liver cells (Liver: Organic Anion Transport Polypeptide- OATP) • **Conjugation in liver cells** (UDP glucuronyl transferase located in smooth endoplasmic reticulum acts on the bilirubin to form bilirubin-diglucuronide (BiliG) which is H2O soluble) • BiliG is actively transported to biliary canaliculi, bile ducts and then to intestine. (small amounts of BiliG and free Bilirubin leak into the circulation.) • **Intestinal phase:** Intestinal bacteria acts on the BiliG to form unconjugated bilirubin and urobilinogen. These are excreted via the gut. • **Enterohepatic circulation**: unconjugated Bilirubin and urobilinogen can re-enter the portal circulation. • Urobilinogen may enter the general circulation to be excreted by the kidneys.	Bold to pass
	c) Describe the composition of bile.	• 97% water • bile pigments (conjugated bilirubin + biliverdin) • bile salts (cholic acid, chenodeoxycholic acid, deoxycholic acid, lithocholic acid) • inorganic salts others: cholesterol, fatty acids, lecithin, fat	3 to pass

Bilirubin 2016-1-A

Stem: Moving onto Physiology.			
Question 4 Bilirubin metabolism / jaundice **Subject:** Phys LOA: 1	1) Describe the metabolism of bilirubin	**Formed from breakdown of Hb** **Bound to albumin** Free bilirubin enters liver cells via OATP family (organic anion transporting polypeptide) binds to cytoplasmic proteins **Conjugated** by glucuronyl-transferase in ER with glucuronic acid to H2O soluble bilirubin diglucuronide Bilirubin diglucuronide actively transported against conc gradient by MDRP-2 to bile caniliculi; small amount escapes into blood, bound to albumin, excreted in urine Intestinal mucosa relatively impermeable to conj bilirubin, gut bacteria convert most to urobilinogens **Enterohepatic circulation:** Some reabsorbed in portal circulation and resecreted. Small amt urobilinogens excreted in urine and faeces (uro and stercobilinogens)	Bold plus one more cause
	2) What are the causes of jaundice?	**Excess production of bilirubin (eg haemolytic anaemia)** Decreased uptake of bilirubin into hepatic cells Disturbed intracellular protein binding or conjugation Disturbed secretion of conjugated bilirubin into the bile cannniliculi **Intra or extrahepatic bile duct obstruction** (1st three liberate free bilirubin; last 2 result in elevated conjugated bilirubin in blood)	Bold to pass

Bilirubin 2012-2

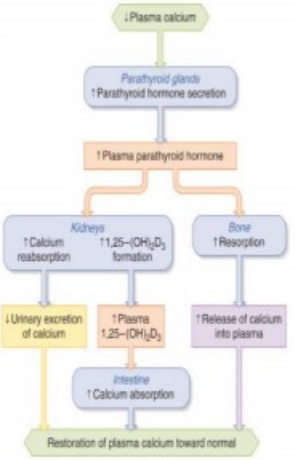
Question 5 LOA: 2	Describe the metabolism and excretion of bilirubin ? What are the causes of jaundice ?	**1)** a) Bilirubin **ex breakdown of Hb**. Bound to **albumin** in circulation. b) Most dissociates in liver, **enters liver cells** as free bilirubin via organic anion transporting polypeptide (OATP), bound to cytoplasmic proteins; c) **conjugated to glucuronic acid via glucuronyl transferase** in smooth ER to form water soluble bilirubin diglucuronide; d) transported against conc gradient into bile canaliculi; **excreted in bile into intestine** Small amount of bilirubin diglucuronide escapes into blood where loosely bound to albumin and excreted in urine. Total plasma bilirubin includes free bili plus small amount conjugated bili. e) **Intestinal bacteria – convert conj bili to urobilinogen** which can be absorbed by the intestinal mucosa , reabsorbed into portal circulation; some re-excreted into bile, some enters general circulation and excreted in urine **2)** a) excess bilirubin production – **haemolysis** b) **decreased uptake bilirubin into cells** c) **disturbed intracellular protein binding or conjugation** d) **disturbed secretion of conjugated bilirubin into the bile cannaliculi** e) intra- or extra-hepatic **bile duct obstruction**. (First 3 liberate free bilirubin, latter 2 cause elevated conjugated bilirubin in plasma)	1) Conjugation + 3 more bolded processes to pass 2. Haemolysis, obstruction + 1 more

Bilirubin 2006-1

TOPIC: bilirubin NUMBER: 5

OPENING QUESTION	Describe the metabolism of bilirubin.	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Breakdown of haemoglobin leads to bilirubin which is bound to albumin in the circulation. In the liver it dissociates and free bilirubin enters the hepatic cell where it is conjugated by glucuronyl transferase with 2 molecules of uridine diphosphoglucuronic acid (UDPGA) to form bilirubin diglucuronide and UDP. The diglucuronide is more water soluble than free bilirubin.		*Must be able to identify metabolism in the liver by glucuronidisation to a more soluble form which is then mostly excreted via bile*
THIRD QUESTION (if needed)	How is bilirubin excreted?		
POINTS REQUIRED	It is mostly passed into the bile ducts and excreted via the intestines. A small amount enters the blood and is measurable as conjugated bilirubin in the blood.		

Calcium Metabolism 2017-2-D

Stem: Moving on to Physiology. Her calcium level was checked.			
Question 5 Calcium haemostasis **Subject**: Physiology LOA: 2 *Ganong. 25th edition. Chapter 21 Hormonal control of Calcium and phosphate metabolism and the physiology of bone*	(a) How does the body regulate Plasma Calcium?	**1,25 – dihydrocholecalciferol** (DHCC) (from Vit D) increases Ca absorption from GIT and kidneys. **PTH** mobilises Ca from bone, increases Ca reabsorption in kidneys, increases 1,25 DHCC formation in kidneys. **Calcitonin** (from thyroid) inhibits bone resorption, increases Ca excretion in urine.	Mention Vit D and PTH PLUS correct direction of effect on Ca to pass
	(b) How is the synthesis of 1,25 – dihydrocholecalciferol (DHCC) (Vit D) regulated?	1,25 –DHCC formed in kidneys by 1alpha-hydroxylase. **Low Ca increases PTH** which stimulates 1alpha-hydroxylase **and increases 1,25 DHCC formation** Low PO4 directly stimulates 1alpha-hydroxylase **High Ca**/high PO4 **inhibits 1,25-DHCC** (increases inactive 24,25-DHCC instead).	b. concept in bold to pass 

Calcium Metabolism 2015-2-C

Stem: Moving on to Physiology.			
Question 5 Calcium metabolism **Subject:** Phys LOA: 1	1. How is plasma calcium regulated?	**1,25-dihydroxycholecalciferol (from Vit D) incr Ca absorption from gut & kidneys.** **PTH mobilises Ca from bone**, incr Ca reabs in kidneys, incr 1,25 DHCC formation in kidneys **Calcitonin** (from thyroid) **inhibits bone resorption**, incr Ca excretion in urine.	2/3 to pass
	2. How is the synthesis of 1,25-dihydroxycholecalciferol (vit D) regulated?	**1,25-DHCC formed in kidneys by 1α-hydroxylase.** **High Ca/high PO4 inh 1,25-DHCC** (incr inactive 24,25-DHCC instead). **Low Ca incr PTH which stimulates 1α-hydroxylase** (low PO4 directly stimulates 1α-hydroxylase).	Bold to pass

Digestion of Carbohydrates 2003-2

TOPIC: Digestion and Absorption of Carbohydrates _____ **NUMBER:** _____ 1a

OPENING QUESTION	Describe the enzymes required for the digestion of carbohydrates and their location	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Salivary Amylase	1	
	2 Duodenum – Pancreatic Amylase	2	
	3 Brush Border Oligosaccharidases	3	
	4 Examples of these oligosaccharidases are: alpha-dextrinase (isomaltase), lactase, sucrase, maltase & trehalase	4	1,2,3 to pass
	5 Final oligosaccharides: alpha-dextrins, maltotriose, maltose, trehalose, lactose, sucrose are metabolized to one of the hexoses (monosaccharides-galactose, fructose or glucose)	5	Bonus if describe how the final monosaccharides produced
	6	6	
SECOND QUESTION (if needed)	Please describe how carbohydrates are absorbed from the gastrointestinal tract.		
POINTS REQUIRED	1 Two phases: first into intestinal mucosal cell and second into interstitial fluid (ECF) and thus into capillaries & portal blood	1	
	2 Glucose/Galactose "secondary active transport" with sodium - low concentration of Na inhibits transport (co-transporters: SGLT-1 & SGLT-2 - sodium-dependent glucose transporter)	2 how does glucose move into the mucosal cells	Bonus if extra detail re co-transporter protein
	3 Glucose/Galactose – "facilitated diffusion" into ICF by GLUT-2	3	
	4 Fructose – "facilitated diffusion" from intestinal lumen by GLUT-5, thence GLUT-2 into ICF	4	
	5 Ribose/Deoxyribose Diffusion	5	
THIRD QUESTION (if needed)			
POINTS REQUIRED	1	1	
	2	2	
	3		

Digestion of Lipids 2003-2

TOPIC: Digestion and Absorption of Lipids_____ **NUMBER:** _____ 1c

OPENING QUESTION	Describe the enzymes required for the digestion of lipids and their location	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Lingual lipase (Ebner's Gland) - active in the stomach on triglycerides	1	2 of 4 to pass
	2 Pancreatic lipase - requires colipase for maximal activity (triglycerides)	2	
	3 Pancreatic bile-salt activated lipase (not only triglycerides but also cholesterol esters, some vitamins & phospholipids)	3	
	4 Cholesteryl ester hydrolase (cholesterol)	4	
	5	5	
	6	6	
SECOND QUESTION (if needed)	What other process is involved in the digestion of lipids?		2 of 3 to pass
POINTS REQUIRED	1 Emulsification	1	
	2 Micelles - formed from bile salts, lecithin and monoglycerides surrounding fatty acids, monoglycerides & cholesterol	2	
	3 Transport lipids thru "unstirred layer" to brush border of mucosal cells	3	
	4	4	
THIRD QUESTION (optional)	Please describe how lipids are absorbed from the gastrointestinal tract.		Bonus marks
POINTS REQUIRED	1 Two phases: first into intestinal mucosal cell and second into interstitial fluid (ECF) and thus into capillaries & portal blood (FFA - free fatty acids) or into lymphatics (chylomicrons)	1	
	2 Into enterocytes: passive diffusion & carriers	2	
	3 Out of enterocytes: depending on size (< 10-12 carbons - directly into portal blood (FFA's) OR > 10-12 carbons - reesterified to triglycerides or cholesteryl esters & packaged in chylomicrons (coating of protein, cholesterol & phospholipids)		

Digestion of Proteins 2006-2

TOPIC: Protein Digestion and Absorption _____ **NUMBER: 5** _____

OPENING QUESTION	Describe how proteins are digested in the gastrointestinal tract?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Stomach - pepsinogens are activated by the gastric acid to produce pepsins and these cleave bond bt amino acids	1	Required for pass
	2 Small intestine - Powerful proteolytic enzymes from pancreas and intestinal mucosa.	2	Additional information
	3 Endopeptidases and exopeptidase hydrolyse amino acids	3	Additional information
SECOND QUESTION (if needed)	How are proteins absorbed from the gastrointestinal tract?		
POINTS REQUIRED	1 Two phases 1. mucosal cell and 2. interstitial fluid and then into capillaries and portal blood	1	Pass = 1 & 2
	2 Seven transport system: Five require Na and two Na independent (Na similar to glucose transport)	2	
	3 Absorption is rapid in duodenum and jejunum and slow in ileum	3	
	4	4	
	5	5	
	6	6	
	7		
THIRD QUESTION (if needed)	How does protein absorption and digestion differ in infants and young children compared to adult?		Pass = 1/2
POINTS REQUIRED	1 Infants absorb more undigested protein	1	
	2 Results in more food allergy but passive immunity	2 Prompt if need be	
	3	3	
	4	4	
	5	5	
	6	6	

Digestion of Proteins 2003-2

TOPIC: Digestion and Absorption of Proteins _____ **NUMBER:** _____ 1b

OPENING QUESTION	Describe the enzymes required for the digestion of proteins and their location	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 Stomach - pepsinogens activated by gastric hydrochloric acid (pH 1.6-3.2) to pepsins result in polypeptides	1	3 of 5 to pass
	2 Small intestine lumen (pH 6.5) - proteolytic enzymes of the pancreas & intestinal mucosa	2	
	3 Examples: endopeptidases (trypsin, chymotrypsin & elastase) & exopeptidases to amino acids	3	
	4 Brush border: (amino, carboxy, endo & di) peptidases to amino acids	4	
	5 Cytoplasm of mucosal cells: after absorption by active transport	5	
	6	6	
	7	7	
	8		
SECOND QUESTION (if needed)	Describe how proteins are absorbed from the gastrointestinal tract.		
POINTS REQUIRED	1 Two phases: first into intestinal mucosal cell and second into interstitial fluid (ICF) and thus into capillaries & portal blood	1	2 of 4 to pass
	2 Into enterocytes: seven different transport systems for amino acids (sodium dependent and independent)	2	
	3 Out of enterocytes: five different transport systems	3	
	4	4	
	5	5	
THIRD QUESTION (if needed)			
POINTS REQUIRED	1	1	
	2	2	
	3		

Exocrine Pancreas 2013-1

Question 5 LOA: 1 Exocrine pancreas	a. List the enzymes secreted from the exocrine pancreas. b. Give at least 3 examples of substrates that these enzymes work on.	Trypsin – proteins,polypeptides Chymotrypsins– proteins,polypeptides Elastase –elastin and some proteins Carboxypeptidase A - proteins,polypeptides Carboxypeptidase B - proteins,polypeptides Colipase –fat droplets Pancreatic Lipase -triglycerides Bile salt –acid lipase –cholesterol esters Cholesterol ester hydrolase–cholesterol esters Pancreatic alpha amylase -starch Ribonuclease -RNA Deoxyribonuclease -DNA Phospholipase A2 –phospholipds	Lipase and at least 2 examples & matched substrates

Exocrine Pancreas 2010-1

5 a) Name the principal pancreatic enzymes and the substances upon which they act.	• Trypsin – proteins, polypeptides • Chymotrypsin – proteins, polypeptides • Elastase – elastin and some other proteins • Carboxypeptidase A & B – proteins, polypeptides • Colipase – fat droplets • Pancreatic lipase – triglycerides • Bile salt-acid lipase – cholesterol esters • Pancreatic α-amylase – starch • Ribonuclease – RNA • Deoxyribonuclease – DNA • Phospholipase A2 – Phospholipids	Must be able to give trypsin, lipase, amylase plus 1 other & their appropriate substrate group (protein, fat, carbohydrate)
5 b) Describe the regulation of pancreatic juice secretion	• Primarily under hormonal control • Secretin acts on the duct to cause production of copious amounts of very alkaline pancreatic juice poor in enzymes. • As flow of pancreatic juice increases it becomes more alkaline because exchange of HCO_3^- for Cl^- in the distal duct is inversely proportional to flow • CCK acts on acinar cells to cause of release of zymogen granules and pancreatic juice rich in enzymes • Acetylcholine also stimulates release of zymogen granules (minor effect ?basis of vagally-mediated pancreatic juice secretion in response to sight/smell of food). Prompt: Do you know any hormones involved in secretion of pancreatic juice?	Must give Secretin and CCK and know that secretin causes mainly alkaline fluid and CCK mainly enzymes.

Exocrine Pancreas 2005-2

1.5 Exocrine function of the pancreas	Describe the composition of pancreatic juice Describe the regulation of secretion of pancreatic juice	Composition: **cations, anions, HCO3-, Digestive enzymes** = Proenzyme trypsinogen converted to trypsin by enteropeptidase (enterokinase) from brush border. Trypsin converts chymotrypsinogens, proelastase, procarboxypeptidases to active enzymes. Digestive enzymes in zymogen granules in acinar cells in alveolar glands, discharged by exocytosis into pancreatic ducts. **Secretin – HCO3-**, 1500ml/day; bile secretion. **CCK – releases zymogen granules** (also vagal acetylcholine)	

Gastric Secretion 2006-1

TOPIC: Regulation of Gastric secretion **NUMBER: 5**

OPENING QUESTION	What factors regulate gastric secretion?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	Neural and hormonal OR Cephalic, Gastric and intestinal Cephalic; food in mouth →vagus, psychologic states eg anger hostility → hypersecretion Gastric: food in stomach, local receptors eg to amino acid and protein digestions→ post ganglionic neurons→ parietal cells→ acid secretion Intestinal: fats, carbohydrates, and acid in duodenum inhibit gastric acid secretion and pepsin secretion as well as motility by neural and hormonal mechanisms Eg peptide YY Neural: Vagal increases gastrin secretion in G cells by GRP. Gastrin stimulates gastric acid and pepsin secretion as well as motility. Hypoglycaemia via vagus to stimulate acid and pepsin secretion Also alcohol and caffeine stimulate gastric secretion		Need to name both and give an example of each (vagus, hormonal eg: gastrin).

Glucose 2016-2-A

Stem: Moving onto Physiology.

TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 2: Glucose homeostasis **Subject:** Phys: LOA: 1	1) What factors influence glucose homeostasis?	Glucose absorption of the **intestine** Glucose **uptake** from the **periphery** – muscle, brain, fat, RBC's and **liver**. Reabsorption in the **kidney** **Gluconeogenesis in liver** – actions of insulin & glucagon.	Name 3 mechanisms for a pass.
	2) What happens to glucose homeostasis in the absence of insulin? *Prompt if only give hyperglycaemia -By what mechanisms does this occur?*	Hyperglycemia due to: 1) Decreasing peripheral uptake of glucose into muscle and fat (direct effect). 2) Reduced glucose uptake by the liver (indirect effect) 3) Increased glucose output by the liver & lack of glycogen synthesis. (GIT, Renal, Brain, RBC uptake remains unaffected.)	For a pass name 2/3 consequences.
	3) By what mechanism does glucose cause the release of insulin?	Specific GLUT 2 transporter in beta cells of the pancreas, converted to pyruvate, metabolized to glutamate via citric acid cycle (CAC) which primes insulin granules for release. Production of ATP also triggers (via K efflux) Ca influx which causes granules to be released.	Basic concept to pass.

Iron Absorption 2016-2-A

Question 4 Iron metabolism and haemoglobin **Subject:** Physiology LOA: 1	How is Iron absorbed from the gastro-intestinal tract?	• **Gastric acid** aids reduction **Fe 3+ to Fe2+** (ferrous) & formation of soluble complexes • **Duodenum= major site of absorption** • **Fe3+ converted to Fe 2+ by ferric reductase** • **Fe2+ transported into enterocytes via** apical membrane iron **transporter** (DMT1) • **Dietary Heme** transported into enterocyte by Heme Transporter (HT) • Heme oxidase releases Fe 2+ from heme • Some intracellular Fe 2+ converted to Fe 3+ & bound to ferritin • Remainder binds to basolateral Fe 2+ transporter ferroportin (FP) and transported to interstitial fluid aided by hephaestin (Hp)	**Pass Criteria: 2 Bold** + 1 other
	What factors reduce Iron absorption from the gastro-intestinal tract? (Hint- what foods, medications or procedures may result in reduced Iron absorption?)	**Dietary**-Phytic acid (cereals), oxalates & phosphates bind Fe to produce insoluble compounds **Surgical**- Partial Gastrectomy (decreased acid)/ duodenal surgery-loss or illnesses (ulcers, sprue) **Physiological**- high iron stores, high recent FE diet, amount of erythropoiesis **Drugs**-antacids, acid lowering, some antibiotics	**Pass Criteria: 1 Bold**
	How is Iron transported in the plasma?	Fe 2+ converted to Fe 3+ & bound to **transferrin**	**Pass Criteria: 1 Bold**

Iron Absorption 2008-1

2.5 Iron Ganong pp 474-478	Please describe how ingested iron is absorbed.	• Most ingested iron is ferric (3+) but the ferrous (2+) form is absorbed. • Minimal absorption in stomach but gastric secretions dissolve iron and aid conversion to the ferrous form. • Almost all absorption in duodenum. Iron is transported into enterocytes via DMT1. • Some stored as ferritin. • Remainder transported out via ferroportin 1 (basolateral transporter) in the presence of hephaestin. Then converted to ferric form and bound to transferrin. • Dietary heme is absorbed by an apical transporter andiron is removed from the porphyrin in cytoplasm.	
	What are the mechanisms that regulate iron absorption?	Precise mechanisms uncertain, probably related to: • Recent dietary intake of iron. • State of body iron stores. • State of erythropoiesis in bone marrow. • The regulatory mechanisms are unclear.	/2

Iron Absorption 2004-2

TOPIC: Iron metabolism _____ **NUMBER:** _____

OPENING QUESTION	How is iron absorbed from the gastro-intestinal tract?	**PROMPTS**	**COMMENTS**
POINTS REQUIRED	1 As heme iron (bound) or free iron	1 Factors?	
	2 Heme iron absorption independent of pH	2	
	3 More Fe^{2+} (soluble form) with gastric acid	3	
	4 Affected by other gut contents	4	
	5 In small bowel	5	
	6 Binds to apoferritin	6	
	7 Transported to portal circulation	7	
	8 Feedback alters rate of absorption		
SECOND QUESTION (if needed)	How is iron transported?		
POINTS REQUIRED	1 Free Fe^{2+} bound to transferrin	1	
	2 To liver then bone marrow	2	
	3	3	
	4	4	
	5	5	
	6	6	
	7		
THIRD QUESTION (if needed)	Physiologically, how is iron lost from the body?		
POINTS REQUIRED	1 Gut cells	1	
	2 Menstruation	2	
	3	3	
	4	4	
	5	5	
	6	6	
	7		

Liver Metabolic Functions 2014-1-D

Stem: Moving onto **PHYSIOLOGY**			
Question 4 Liver metabolic functions especially bilirubin metabolism. **Subject:** Phys LOA: 1	(a) List the principal functions of the liver	(a) **Bile formation** (500ml/day) • **Synthesis** – protein, coag factors, albumin • **Inactivation** / detoxification – drugs, toxins, active circulating substances • Nutrient vitamin **absorption, metabolism / control** (e.g. glucostat), AAs, lipids, fat sol vitamins • **Immunity** (esp. gut organisms) – Kupffer / macrophages in sinusoid endothelium	(a) 3/5 bold with an example to pass
	(b) Describe the metabolism of bilirubin.	(b) • **Formed by breakdown of haeme, Hb** • Bound to albumin • In liver – actively transported (OATP) as dissociates – binds to cytoplasmic proteins • **Conjugated by gluc-transferase in ER with glucuronic acid to H2O sol bil-digluc** • Bil di gluc active transport (MDRP2) against gdt to bile canaliculi – to gut (<5% bil/bdg reflux to blood) • Intestinal mucosa relatively impermeable • Gut bacteria act / convert most to urobilinogens • **Some bile pigments / urobilinogens/unconj bil reabsorbed in portal circulation – most resecreted = enterohepatic circulation** • Small amounts urobil in blood excreted in urine – urobilinogen and faeces – stercobi	(b) Bold to pass

Liver Metabolic Functions 2009-2

Question 4:	**What are the principal functions of the Liver.**	**1) Bile formation (500 mls a day) - Excretion, elimination,** digestion **2) Synthesis- protein/** coag/ biding prot/alb **3) Inactivation/ detox –**drugs/toxins/ active circ substances **4) Nutrient vitamin absorption, metabolism/ control (e.g. glucostat)** AAs, lipids, fat sol vits etc **5) Immunity (partic gut orgs)**- Kupffer/ Macrophages in sinusoid endothelium	3/5 named functions (or part of function eg some idea)
b)	**Describe bilirubins path from production to excretion?**	1. **Most formed by breakdown of Heme /Hb.** 2. **Bilirubin bound to albumin *** 3. **In liver actively transported (OATP) as dissociates – binds to cytoplasmic proteins.** 4. **Conjugated by gluc-transferase (*in ER) with glucoronic acid to H20 sol bil-digluc** 5. **Bil di gluc active transport (MDRP2) against gdt to bile canaliculi – to gut. (<5% bil/bdg reflux to blood)** 6. **Intestinal mucosae relatively impermeable** 7. **Gut bacteria act / convert most to urobilinogens*** 8. **Some bile pigments/ urobilinogens/unconj bil reabsorbed in portal circn –most resecreted– entero hepatic circulation.** 9. **Small amounts urobil in blood excreted in urine – urobilinogen and faeces – stercobil.**	Pass Fail 4 elements in proper order/ prompt if stuck on excessive detail e.g. just a general overview of production to excretion. Pass does not require this detail!

Water and Electrolyte Absorption 2013-1

Question 5 GIT handling of water and electrolytes LOA: 1	a. Explain the mechanisms of absorption of water and electrolytes in the gastrointestinal tract. Prompt: How is sodium absorbed?	**Absorption:** After meals – **fluid reuptake due to coupled transport of nutrients**, e.g. glucose and Na (Water reabsorbed 8800 ml) Between meals – NaCl enters across the apical membrane via the coupled activity of a Na/H exchanger and a Cl/HCO3 exchanger (electroneutral mechanism in small intestine & colon). In distal colon, Na enters the epithelial cell via epithelial Na Channels (electrogenic mechanism).	Bold and 1 mechanism of Na absorption somewhere
	b. Explain the mechanisms of water and electrolyte secretion in the gastrointestinal tract. Prompt: How is chloride secreted?	**Secretion:** **Cl secretion occurs continuously in the small intestine & colon**. Cl uptake occurs via Na/K/2Cl co-transporter and is secreted into the lumen via Cl channels (CFTR = cystic fibrosis transmembrane conductance regulator). **Water endogenous secretions** 7000 ml	Bold and 1 mechanism of Cl secretion somewhere Note: Water balance Input: Ingested 2000 ml & Endogenous secretions 7000 ml; Output: Reabsorbed 8800 ml; Balance in stools 200 ml