DOWNLOAD PDF INTESTINAL ABSORPTION

Chapter 1 : Digestion and Absorption of Fats

intestinal absorption the transfer of the products of digestion, minerals and water (also drugs) from the intestine into the blood or lymph. Food products are absorbed from the small intestine, via its lining of enterocytes (where some further digestive processes take place); hexoses from carbohydrates, and amino acids and peptides from proteins, enter surrounding blood vessels, thence in the.

Absorption refers to the movement of nutrients, water and electrolytes from the lumen of the small intestine into the cell, then into the blood. In this article, we will look at the digestion and absorption of carbohydrates, protein and lipids. Digestion of starch is initiated in the mouth, facilitated by salivary amylase. The majority of carbohydrate digestion occurs in the stomach and duodenum. Disaccharides occurring naturally in food do not require amylase to break them down. Brush border enzymes lactase, sucrase, trehalase hydrolyse these compounds into molecules of glucose, galactose and fructose. The process of digestion is completed in the small intestine with brush border and pancreatic enzymes. They split the oligopeptides into amino acids, dipeptides and tripeptides. Absorption Amino acids are absorbed via a Sodium cotransporter, in a similar mechanism to the monosaccharides. Lipids Digestion Lipids are hydrophobic, and thus are poorly soluble in the aqueous environment of the digestive tract. The remainder of the lipids are digested in the small intestine. Absorption The products from digestion are released at the apical membrane and diffuse into the enterocyte. The chylomicrons are too large to enter circulation, so they enter lymphatic system via lacteals. Frank Boumphrey, MD derivative work: Hazmat2 This file was derived from Bile1. By enveloping the lipid, the bile enhances absorption. Clinical Relevance â€" Steatorrhea Steatorrhea is due to a disruption to the normal absorption of lipids, leading to fat filled faeces. There are numerous underlying causes for this such as pancreatitis, which prevents the correct secretion of pancreatic lipase and so lipids remain undigested. Another cause is gallstones which prevent bile from entering the duodenum and again prevents maximal absorption of lipids. However, the absorption in the small intestine can be compromised, such as in inflammatory bowel diseases. To distinguish between the underlying causes of Steatorrhea, the small intestine and biliary tree must be visualised. The small intestine can be visualised via endoscopy or radiography whilst the biliary tree can be visualised with endoscopic retrograde cholangiopancreatography.

Chapter 2 : What Is Malabsorption? - Absorption Disorders - calendrierdelascience.com

The small intestine or small bowel is the part of the gastrointestinal tract between the stomach and the large intestine, and is where most of the end absorption of food takes place. The small intestine has three distinct regions - the duodenum, jejunum, and ileum.

Intestinal absorption of iron figure 1 Iron absorption occurs in two steps Absorption of iron into the enterocyte at the luminal surface of the enterocytes Transport of iron to the lamina propria at the basilateral surface of the enterocyte click here for fig 1 Absorption of iron into the enterocyte Dietary iron is in two forms, heme and non-heme. Plant foods contain only non-heme iron. Most of the iron available for absorption comes from non-meat sources even in meat eaters. The widely held perception that vegetarian diets contain less iron than meat eaters is not true. Vegetarian foods have less heme iron. Different pathways absorb heme and non-heme iron. A receptor for the absorption of heme has been postulated but not identified. Once within the cell the haemin is acted upon by heme oxygenase 1 to release the iron. The heme iron is added to the enterocyte iron pool and follows the same path out of the cell as non-heme iron. Food does not affect the absorption of heme iron. Some heme may be absorbed directly and is bound to hemoprixin. Non-Heme Iron Dietary non-Heme iron is present in the ferric state. Ferric iron is insoluble and needs to be converted to the more soluble ferrous state for absorption. The reduction of ferric iron is aided by the acidic environment of the stomach, dietary components like ascorbic acid and duodenal cytochrome b Dcytb. Dcytb does not appear to be essential for iron absorption. Ferrous iron is absorbed by the divalent metal ion transporter DMT1. DMT1 is expressed at the brush-border of the enterocyte near the tips of small intestinal villi. DMT1 needs protons to co-transport with iron. Protons come from the gastric juice and are most abundant in the duodenum. Most of the iron is absorbed in the duodenum. Antacid, H2 antagonists and proton pump inhibitors hamper iron absorption by reducing hydrogen ion availability. All these drugs have been shown to impair the efficacy of medicinal iron. The absorption of non-heme iron, unlike heme iron, is affected by food. Foods enhancing iron absorption: Ascorbate, animal proteins, human milk, keto sugars, organics, amino acids that form soluble chelates with iron enhance absorption of non-heme iron. Transport of iron to the lamina propria Iron is transported to the lamina propria by an iron transporter ferroportin. Ferroportin transports iron in the ferrous form. This needs to be oxidized to the ferric form for binding to transferrin. Hephaestin oxidizes ferrous iron to ferric iron. Ferroportin is the key to controlling body iron. In iron deficient state ferroportin expression at the basolateral surface of the enterocyte is increased and more iron is transferred to the blood. When the body is iron repleted ferroportin expression is low and the iron remains in the enterocyte as ferritin. The liver, on sensing adequate iron stores, secretes a peptide hepcidin that binds to ferroportin. Binding of ferroportin to hepcidin causes internalisation and degradation of ferroportin preventing iron transport.

Chapter 3 : Human Intestinal Absorption | Drugs absorbed from the intestine into the bloodstream

The large intestine, or colon, has several roles including water absorption and immunity. The chyme that enters the colon is already very concentrated as most of the water has already been absorbed.

The chyme that enters the colon is already very concentrated as most of the water has already been absorbed. Thus, the large intestine is specialised to work in the environment this produces. In this article, we will review the functions of the intestine and how they are achieved. These protect the intestinal wall from the plethora of anaerobic bacteria in the colon and from the pressure exerted on the walls by the concentrated chyme soon to become faeces. As the chyme is very concentrated by the time it reaches here, the colon must work against a larger osmotic pressure gradient than in the rest of the GIT. In other words, it must move water against the gradient for osmosis. The colon also helps to transport ions. Sodium â€" this ion may be absorbed by various methods: Sodium-hydrogen antiporter on the luminal membrane Epithelial sodium channels Enhanced by absorption of short-chain fatty acids in the colon via specialised symporters Chloride and bicarbonate â€" the movement of sodium into the plasma produces an electrochemical gradient to allow absorption of chloride. Chloride ions are exchanged for bicarbonate ions causing net bicarbonate secretion. Water â€" the absorption of these electrolytes creates an osmotic gradient to allow further absorption of water. Potassium â€" absorption of water along the length of the bowel concentrates potassium in the lumen. This provides an electrochemical gradient for the movement of potassium into the plasma. In the colon potassium may be absorbed or secreted depending on the remaining concentration in the lumen and the electrochemical gradient created by the active absorption of sodium. Secretion usually occurs when the luminal concentration of potassium ions is below 25mM. Vitamins and fats â€" short-chain fatty acids, crucial B vitamins such as B6 and B12 and vitamin K required for blood clotting are produced by the digestion of chyme by the commensal microbial flora of the colon. Regulation of Absorption Absorption in the gastrointestinal tract is regulated by neuroendocrine mechanisms. In the colon endocrine mechanisms used include: Aldosterone â€" increases the net absorption of water and electrolytes by stimulating the basolateral sodium-potassium ATP-ase. This increases the electrochemical gradient and driving force for sodium absorption. It also increases transcription of epithelial sodium channels. Glucocorticoids and somatostatin â€" act to increase water and electrolyte absorption by increasing the action of the basolateral sodium-potassium ATP-ase. The intestines are innervated by the enteric nervous system whose: Parasympathetic innervation promotes net secretion from the intestines Sympathetic innervation promotes net absorption from the intestines.

Chapter 4 : Differences in Small & Large Intestines | Children's Pittsburgh

The small intestine is the part of the gastrointestinal tract between the stomach and the large intestine where much of the digestion of food takes place. The primary function of the small intestine is the absorption of nutrients and minerals found in food.

Development of the digestive system The small intestine develops from the midgut of the primitive gut tube. The loop grows so fast in length that it outgrows the abdomen and protrudes through the umbilicus. By week 10, the loop retracts back into the abdomen. Between weeks six and ten the small intestine rotates anticlockwise, as viewed from the front of the embryo. It rotates a further degrees after it has moved back into the abdomen. This process creates the twisted shape of the large intestine. Digestion[edit] The small intestine is where most chemical digestion takes place. Many of the digestive enzymes that act in the small intestine are secreted by the pancreas and liver and enter the small intestine via the pancreatic duct. Pancreatic enzymes and bile from the gallbladder enter the small intestine in response to the hormone cholecystokinin , which is produced in the small intestine in response to the presence of nutrients. Secretin , another hormone produced in the small intestine, causes additional effects on the pancreas, where it promotes the release of bicarbonate into the duodenum in order to neutralize the potentially harmful acid coming from the stomach. The three major classes of nutrients that undergo digestion are proteins , lipids fats and carbohydrates: Proteins are degraded into small peptides and amino acids before absorption. Proteolytic enzymes, including trypsin and chymotrypsin , are secreted by the pancreas and cleave proteins into smaller peptides. Carboxypeptidase, which is a pancreatic brush border enzyme, splits one amino acid at a time. Aminopeptidase and dipeptidase free the end amino acid products. Lipids fats are degraded into fatty acids and glycerol. Pancreatic lipase breaks down triglycerides into free fatty acids and monoglycerides. Pancreatic lipase works with the help of the salts from the bile secreted by the liver and stored in the gall bladder. Bile salts attach to triglycerides to help emulsify them, which aids access by pancreatic lipase. This occurs because the lipase is water-soluble but the fatty triglycerides are hydrophobic and tend to orient towards each other and away from the watery intestinal surroundings. The bile salts emulsify the triglycerides in the watery surroundings until the lipase can break them into the smaller components that are able to enter the villi for absorption. Some carbohydrates are degraded into simple sugars, or monosaccharides e. Pancreatic amylase breaks down some carbohydrates notably starch into oligosaccharides. Other carbohydrates pass undigested into the large intestine and further handling by intestinal bacteria. Brush border enzymes take over from there. The most important brush border enzymes are dextrinase and glucoamylase, which further break down oligosaccharides. Other brush border enzymes are maltase, sucrase and lactase. Lactase is absent in some adult humans and, for them, lactose a disaccharide , as well as most polysaccharides, is not digested in the small intestine. Some carbohydrates, such as cellulose , are not digested at all, despite being made of multiple glucose units. This is because the cellulose is made out of beta-glucose, making the inter-monosaccharidal bindings different from the ones present in starch, which consists of alpha-glucose. Humans lack the enzyme for splitting the beta-glucose-bonds, something reserved for herbivores and bacteria from the large intestine. Absorption[edit] Digested food is now able to pass into the blood vessels in the wall of the intestine through either diffusion or active transport. The small intestine is the site where most of the nutrients from ingested food are absorbed. The inner wall, or mucosa, of the small intestine is lined with simple columnar epithelial tissue. Structurally, the mucosa is covered in wrinkles or folds called plicae circulares , which are considered permanent features in the wall of the organ. They are distinct from rugae which are considered non-permanent or temporary allowing for distention and contraction. From the plicae circulares project microscopic finger-like pieces of tissue called villi Latin for "shaggy hair". The individual epithelial cells also have finger-like projections known as microvilli. The functions of the plicae circulares, the villi, and the microvilli are to increase the amount of surface area available for the absorption of nutrients , and to limit the loss of said nutrients to intestinal fauna. Each villus has a network of capillaries and fine lymphatic vessels called lacteals close to its surface. The epithelial cells of the villi transport nutrients from the lumen of the intestine into these capillaries amino acids

and carbohydrates and lacteals lipids. The absorbed substances are transported via the blood vessels to different organs of the body where they are used to build complex substances such as the proteins required by our body. The material that remains undigested and unabsorbed passes into the large intestine. Absorption of the majority of nutrients takes place in the jejunum , with the following notable exceptions: Iron is absorbed in the duodenum. Vitamin B12 and bile salts are absorbed in the terminal ileum. Water is absorbed by osmosis and lipids by passive diffusion throughout the small intestine.

Chapter 5 : Absorption Disorders Overview, Types - Absorption Disorders - calendrierdelascience.com

Intestinal Absorption Physiology The key to our metabolism! Digestion is the set of metabolic mechanical and biochemical processes which transform nutrients into substances that can be assimilated by our bodies.

Print Overview of Absorption Disorders Absorption disorders, also sometimes called malabsorption syndromes, are characterized by problems digesting or absorbing substances called nutrients in the diet. Nutrients include vitamins, minerals, carbohydrates e. Digestion, which occurs in the gastrointestinal GI tract or digestive system, is the process of breaking down food into a form that can be absorbed into the bloodstream and used by the body. The digestive tract consists of the following organs: Mouth Esophagus muscular tube that carries food to the stomach Stomach Large intestine large bowel or colon Biliary system i. The lining of the small intestine called the mucosa contains folds, creases, and finger-like projections called villi and microvilli that increase the surface area of the intestinal lining, providing a larger surface with which to absorb nutrients. The mucosa also contains special cells that increase the absorption of nutrients. Types of Absorption Disorders There are a number of different types of absorption disorders, most of which result in a decreased ability to absorb nutrients i. Common absorption disorders include celiac disease caused by intolerance to gluten, a protein in wheat, barley, and rye and lactose intolerance. Lactose intolerance is an inability to break down a sugar in dairy products lactose. It is caused by a lack of a certain enzyme called lactase that helps convert lactose into a form that can be used by the body i. Lactose intolerance also is called primary or secondary lactase deficiency. Primary lactase deficiency is a common condition that develops over time as the body produces less lactase. This process begins around 2 years of age, but symptoms usually develop much later in life. Secondary lactase deficiency occurs when damage to the small intestine reduces the production of lactase. Conditions that can cause secondary lactase deficiency include celiac disease and inflammatory bowel disease e. Tropical sprue is more common in tropical and subtropical areas of the world, including the Caribbean and Southeast Asia. The exact cause for the condition is unknown, but it may be related to an infection that damages the lining of the small intestine. Symptoms include anemia, diarrhea, weight loss, and malnutrition. Tropical sprue is treated with antibiotics. Treatment involves antibiotics to destroy the bacteria and also may involve intravenous IV; through a vein fluid and electrolyte replacement and dietary supplements. Electrolytes are substances e.

Chapter 6 : Malabsorption - Wikipedia

Human Intestinal Absorption Human Intestinal Absorption Human Intestinal Absorption (HIA) is the process through which orally administered drugs are absorbed from the intestine into the bloodstream.

Foodstuffs typically also contain phospholipids, sterols like cholesterol and many minor lipids, including fat-soluble vitamins. Finally, small intestinal contents contain lipids from sloughed epithelial cells and considerable cholesterol delivered in bile. In order for the triglyceride to be absorbed, two processes must occur: Large aggregates of dietary triglyceride, which are virtually insoluble in an aqueous environment, must be broken down physically and held in suspension - a process called emulsification. Triglyceride molecules must be enzymatically digested to yield monoglyceride and fatty acids, both of which can efficiently diffuse or be transported into the enterocyte The key players in these two transformations are bile acids and pancreatic lipase, both of which are mixed with chyme and act in the lumen of the small intestine. Bile acids are also necessary to solubilize other lipids, including cholesterol. Emulsification, Hydrolysis and Micelle Formation Bile acids play their first critical role in lipid assimilation by promoting emulsification. As derivatives of cholesterol, bile acids have both hydrophilic and hydrophobic domains i. On exposure to a large aggregate of triglyceride, the hydrophobic portions of bile acids intercalate into the lipid, with the hydrophilic domains remaining at the surface. Such coating with bile acids aids in breakdown of large aggregates or droplets into smaller and smaller droplets. Hydrolysis of triglyceride into monoglyceride and free fatty acids is accomplished predominantly by pancreatic lipase. The activity of this enzyme is to clip the fatty acids at positions 1 and 3 of the triglyceride, leaving two free fatty acids and a 2-monoglyceride. The drug orlistat Xenical that is promoted for treatment of obesity works by inhibiting pancreatic lipase, thereby reducing the digestion and absorption of fat in the small intestine. Shortly after a meal, lipase is present within the small intestine in rather huge quantities, but can act only on the surface of triglyeride droplets. For a given volume of lipid, the smaller the droplet size, the greater the surface area, which means more lipase molecules can get to work. As monoglycerides and fatty acids are liberated through the action of lipase, they retain their association with bile acids and complex with other lipids to form structures called micelles. Micelles are essentially small aggregates nm in diameter of mixed lipids and bile acids suspended within the ingesta. As the ingesta is mixed, micelles bump into the brush border of small intestinal enterocytes, and the lipids, including monoglyceride and fatty acids, are taken up into the epithelial cells. Absorption and Transport into Blood The major products of lipid digestion - fatty acids and 2-monoglycerides - enter the enterocyte by simple diffusion across the plasma membrane. A considerable fraction of the fatty acids also enter the enterocyte via a specific fatty acid transporter protein in the membrane. Once inside the enterocyte, fatty acids and monoglyceride are transported into the endoplasmic reticulum, where they are used to synthesize triglyeride. Beginning in the endoplasmic reticulum and continuing in the Golgi, triglyceride is packaged with cholesterol, lipoproteins and other lipids into particles called chylomicrons. Remember where this is occurring - in the absorptive enterocyte of the small intestine. Chylomicrons are extruded from the Golgi into exocytotic vesicles, which are transported to the basolateral aspect of the enterocyte. The vesicles fuse with the plasma membrane and undergo exocytosis, dumping the chylomicrons into the space outside the cells. Because chylomicrons are particles, virtually all steps in this pathway can be visualized using an electron microscope, as the montage of images to the right demonstrates. Transport of lipids into the circulation is also different from what occurs with sugars and amino acids. Instead of being absorbed directly into capillary blood, chylomicrons are transported first into the lymphatic vessel that penetrates into each villus. Chylomicron-rich lymph then drains into the system lymphatic system, which rapidly flows into blood. Blood-borne chylomicrons are rapidly disassembled and their constitutent lipids utilized throughout the body. When large numbers of chylomicrons are being absorbed, the lymph draining from the small intestine appears milky and the lymphatics are easy to see. In the image below, of abdominal contents from a coyote, the fine white lines arrows are intestinal lymphatics packed with chylomicrons. That lymph passes through mesenteric lymph nodes LN and then into larger lymphatics. Another lipid of importance that is absorbed in the small intestine is cholesterol.

Cholesterol homeostatis results from a balance of cholestrol synthesis, absorption of dietary cholesterol, and elimination of cholesterol by excretion in bile. Years ago it was shown that cholesterol, but not plant sterols, is readily absorbed in the intestine. More recently, a specific transport protein NPC1L1 has been identified that ferries cholesterol from the intestinal lumen into the enterocyte. From there, a bulk of the cholesterol is esterified, incorporated into chylomicrons and shuttled into blood by the mechanisms described above. If you are interested in confirming for yourself at least some of the processes described above, you should perform the following experiment: Consume a cup of rich cream or a sack of fast-food French fries. Do something productive like studying for about 30 minutes. Draw a blood sample from yourself a capillary tube is enough - use an anticoagulant to prevent clotting. Centrifuge the blood sample to separate cells and plasma. When you examine your plasma it will look distinctly milky due to the presence of billions of light-reflecting chylomicrons the condition is called lipemia. If you want extra credit, continue the blood sampling every 15 minutes until your plasma clears, then plot your results on graph paper. Alternatively, you can simply examine the image to the right to see what dog serum looks like after several hours of fasting in comparison to lipemic serum collected shortly after a meal of puppy chow.

Chapter 7 : Absorption in the Small Intestine

Alternative names: Impaired Intestinal Absorption This condition is a state of impaired absorption of nutrients in the small calendrierdelascience.comic causes - of which there are many - lead to different patterns in malabsorption, involving for example fat and fat-soluble vitamins (A, D, E and K), vitamin B12, folic acid, iron, protein or carbohydrates.

Diarrhea , weight loss , flatulence , abdominal bloating , abdominal cramps , and pain may be present. Although diarrhea is a common complaint, the character and frequency of stools may vary considerably ranging from over 10 watery stools per day to less than one voluminous putty-like stool, the latter causing some patients to complain of constipation. Not only do unabsorbed nutrients contribute to stool mass but mucosal fluid and electrolyte secretion is also increased in diseases associated with mucosal inflammation such as coeliac disease. In addition, unabsorbed fatty acids, converted to hydroxy-fatty acids by colonic flora, as well as unabsorbed bile acids both impair absorption and induce secretion of water and electrolytes by the colon adding to stool mass. Weight loss is common among patients with significant intestinal malabsorption but must be evaluated in the context of caloric intake. Some patients compensate for fecal wastage of unabsorbed nutrients by significantly increasing their oral intake. Eliciting a careful dietary history from patients with suspected malabsorption is therefore crucial. Excessive flatus and abdominal bloating may reflect excessive gas production due to fermentation of unabsorbed carbohydrate, especially among patients with primary or secondary disaccharidase deficiency. Malabsorption of dietary nutrients and excessive fluid secretion by inflamed small intestine also contribute to abdominal distention and bloating. Prevalence, severity, and character of abdominal pain vary considerably among the various disease processes associated with intestinal malabsorption. For example, there is increasing epidemiologic evidence that more patients with coeliac disease present with anemia and osteopenia in the absence of significant classic gastrointestinal symptoms. Microcytic, macrocytic , or dimorphic anemia may reflect impaired iron , folate, or vitamin B12 absorption. Purpura , subconjunctival hemorrhage , or even frank bleeding may reflect hypoprothrombinemia secondary to vitamin K malabsorption. Osteopenia is common, especially in the presence of steatorrhea. Impaired calcium and vitamin D absorption and chelation of calcium by unabsorbed fatty acids resulting in fecal loss of calcium may all contribute. If calcium deficiency is prolonged, secondary hyperparathyroidism may develop. Prolonged malnutrition may induce amenorrhea, infertility, and impotence. Edema and even ascites may reflect hypoproteinemia associated with protein losing enteropathy caused by lymphatic obstruction or extensive mucosal inflammation. Dermatitis and peripheral neuropathy may be caused by malabsorption of specific vitamins or micronutrients and essential fatty acids. Symptoms can be intestinal or extra-intestinal - the former predominates in severe malabsorption. Diarrhoea , often steatorrhoea , is the most common feature. Watery, diurnal and nocturnal, bulky, frequent stools are the clinical hallmark of overt malabsorption. It is due to impaired water, carbohydrate and electrolyte absorption or irritation from unabsorbed fatty acid. The latter also results in bloating , flatulence and abdominal discomfort. Cramping pain usually suggests obstructive intestinal segment e.

Chapter 8 : Small intestine - Wikipedia

Pregnancy, Lactation, Estrogen and Prolactin and Intestinal Calcium Absorption. Although 1,25(OH) 2 D 3 is the principal hormone regulating active intestinal calcium absorption, other hormones have been shown to influence the process as well.

What is malabsorption syndrome? The main role of your small intestine is to absorb nutrients from the food you eat into your bloodstream. Nutrients that the small intestine often has trouble absorbing can be macronutrients proteins, carbohydrates, and fats , micronutrients vitamins and minerals , or both. Many things can lead to malabsorption syndrome, from certain diseases to infections or birth defects. Possible causes Factors that may cause malabsorption syndrome include: Your stomach may not be able to produce the enzymes it needs to digest certain foods. Or your body may not be able to mix the food you eat with the enzymes and acid produced by your stomach. Rare causes There are also some uncommon disorders that can result in malabsorption. One of these is called short bowel syndrome SBS. With SBS, the small intestine is shortened. This makes the intestine less able to absorb nutrients. SBS may be a birth defect, or it may be caused by surgery. Certain diseases may cause malabsorption. These include tropical sprue , a condition most common in the Caribbean, India, and other parts of Southeast Asia. This disease may be related to environmental factors, such as toxins in food , infection, or parasites. Recognizing the symptoms of malabsorption syndrome Symptoms of malabsorption syndrome are caused when unabsorbed nutrients pass through the digestive tract. Other symptoms are a result of a deficiency of that nutrient, which is caused by its poor absorption. You may have light-colored , foul-smelling stools that are soft and bulky. Stools are difficult to flush and may float or stick to the sides of the toilet bowl. You may have dry hair , hair loss , or fluid retention. Fluid retention is also known as edema and will manifest as swelling. You may have anemia , malnutrition , low blood pressure , weight loss , or muscle wasting. Malabsorption may affect people based on age or gender. For instance, women may stop menstruating , and children may not grow properly. Their weight or rate of weight gain may be significantly below that of other children of a similar age and gender. Another sign of malabsorption in children is that they may avoid certain foods. Risk factors for malabsorption syndrome include: Certain tests are used to confirm the diagnosis. These tests may include: Stool tests Stool tests can measure fat in samples of stool, or feces. These tests are the most reliable because fat is usually present in the stool of someone with malabsorption syndrome. Blood tests These tests measure the level of specific nutrients in your blood, such as vitamin B , vitamin D , folate , iron , calcium , carotene, phosphorus , albumin , and protein. A lack of one of these nutrients may not necessarily mean you have malabsorption syndrome. Normal levels of these nutrients suggest that malabsorption is not the problem. Breath tests Breath tests can be used to test for lactose intolerance. Bacteria in the colon break down the lactose and produce hydrogen gas. The excess hydrogen is absorbed from your intestine, into your bloodstream, and then into your lungs. If you have hydrogen gas in your breath after ingesting a product containing lactose, you may have lactose intolerance. Imaging tests Imaging tests, which take pictures of your digestive system, may be done to look for structural problems. Biopsy You may have a biopsy if your doctor suspects you have abnormal cells in the lining of your small intestine. A biopsy will likely be done using an endoscopy. A tube is inserted into your mouth and sent through your esophagus and stomach and into your small intestine to take a small sample of cells. Treatment options for malabsorption syndrome Your doctor will likely start your treatment by addressing symptoms such as diarrhea. Medications such as loperamide can help. Your doctor will also want to replace the nutrients and fluids that your body has been unable to absorb. And they may monitor you for signs of dehydration , which can include increased thirst , low urine output , and dry mouth , skin , or tongue. Next, your doctor will provide care based on the cause of the absorption problem. At this point, your doctor may refer you to a dietitian. Your dietitian may recommend: Find a great selection of enzyme supplements here. Your dietitian may recommend high doses of vitamins or other nutrients to make up for those that are not being absorbed by your intestine. Your dietitian may adjust your diet to increase or decrease certain foods or nutrients. For instance, you may be advised to avoid foods high in fat to decrease diarrhea, and increase foods

high in potassium to help balance your electrolytes. Your doctor and your dietitian can help create a treatment plan that will manage your malabsorption symptoms and allow your body to obtain the nutrients and fluids it needs to function normally.

Chapter 9 : Intestinal Absorption Physiology | Official web site of the Montignac Method

Malabsorption syndrome refers to a number of disorders in which the small intestine is unable to absorb enough nutrients. your doctor will provide care based on the cause of the absorption.

Digestion and absorption of fats Most of the fat in the human diet is in the form of triacylglycerol TAG , which consists of three fatty acids linked to glycerol. In the digestive tract, TAG is hydrolyzed by the enzyme pancreatic lipase, to release free fatty acids and monoglycerides. Emulsification and digestion The key issue in the digestion and absorption of fats is one of solubility: The digestive enzyme, pancreatic lipase, is water soluble and can only work at the surface of fat globules. Digestion is greatly aided by emulsification, the breaking up of fat globules into much smaller emulsion droplets. Bile salts and phospholipids are amphipathic molecules that are present in the bile. Motility in the small intestine breaks fat globules apart into small droplets that are coated with bile salts and phospholipids, preventing the emulsion droplets from re-associating. The emulsion droplets are where digestion occurs. Emulsification greatly increases the surface area where water-soluble pancreatic lipase can work to digest TAG. Another factor that helps is colipase, an amphipathic protein that binds and anchors pancreatic lipase at the surface of the emulsion droplet. Micelles After digestion, monoglycerides and fatty acids associate with bile salts and phopholipids to form micelles. Micelles are necessary because they transport the poorly soluble monoglycerides and fatty acids to the surface of the enterocyte where they can be absorbed. As well, micelles contain fat soluble vitamins and cholesterol. The figure at right illustrates that micelles are small enough to fall between the the microvilli. Absorption Micelles are constantly breaking down and re-forming, feeding a small pool of monoglycerides and fatty acids that are in solution. Only freely dissolved monoglycerides and fatty acids can be absorbed, NOT the micelles. Because of their nonpolar nature, monoglycerides and fatty acids can just diffuse across the plasma membrane of the enterocyte. Some absorption may be facilitated by specific transport proteins for instance see below, for cholesterol. Chylomicrons Once inside the enterocyte, monoglycerides and fatty acids are re-synthesized into TAG. The TAG is packaged, along with cholesterol and fat soluble vitamins, into chylomicrons. Chylomicrons are lipoproteins, special particles that are designed for the transport of lipids in the circulation. You can review the structure of lipoproteins by visiting the web page on lipoproteins from fall quarter. Chylomicrons are released by exocytosis at the basolateral surface of the enterocytes. Because they are particles, they are too large to enter typical capillaries. Instead they enter lacteals, lymphatic capillaries that poke up into the center of each villus. Chylomicrons then flow into the circulation via lymphatic vessels, which drain into the general circulation at the large veins in the chest. TAG in chylomicrons and other lipoproteins is hydrolyzed by lipoprotein lipase, an enzyme that is found in capillary endothelial cells. Monoglycerides and fatty acids released from digestion of TAG then diffuse into cells. The figure at the right summarizes the various steps involved in fat absorption. Cholesterol absorption Intestinal cholesterol absorption is important because of the clinical relevance of cholesterol: As shown in the figure, some of the cholesterol in the small intestine is dietary cholesterol, and some is put there by the liver, arriving via the bile. Of the total cholesterol that passes through the small intestine, only half is typically absorbed, and the rest is eliminated in the feces. Thus, cholesterol in the bile is an example of a substance that is targeted for excretion via the digestive tract. The drug ezetimibe blocks a protein that specifically mediates cholesterol transport across the apical plasma membrane of enterocytes. Ezetimibe has been shown to be effective at reducing levels of LDL cholesterol, particularly when combined with a statin, a drug that inhibits cholesterol synthesis in the liver. The most recent results of a large clinical trial show that further lowering of LDL cholesterol with a combination of ezetimibe and a statin provides a modest benefit in lowering the risk of myocardial infarction and stroke.