

# [Digestive system of a pig](https://assignbuster.com/digestive-system-of-a-pig/)

### Learning objectives:

After you have studied this chapter, you should:

1. Get a fundamental understanding of the porcine digestive tract
2. Describe the essential digestive processes
3. Understand the role of the digestive organs in digestion and absorption

## 1. Introduction (HNL/MSH)

## 2. Anatomy of the digestive system (HNL)

The anatomy the porcine digestive tract has been described and illustrated in detail by others (e. g. Sisson, 1975, Moran, 1982)[1] and will only be briefly described in the current chapter.

The digestive system of the pig is fundamentally similar to all other monogastric mammals, but the evolutionary development in size and digestive capacity reflects greatly the habitual diet. Pigs are true omnivores but with a large fraction of the diet coming from plant material. As such they have a great capacity to digest enzyme degradable carbohydrates in the upper part of the gastrointestinal tract, and a well-developed ecosystem in the large intestine to partly ferment and utilize fibrous material.

### 2. 1 Mouth and salivary glands

The pig is born with 8 deciduous teeth increasing to 32 with age. The complete set of permanent teeth consists of 44 teeth with 3 pairs of incisors, 1 pair of canines, 4 pair of premolars, and 3 pair of molars, which are usually not fully acquired until 18 months of age[2]. The oral cavity is lined with a simple stratified squamous epithelium, and saliva is mainly secreted from 3 large glands; the parotid glands, the mandibular glands, and the sublingual glands. Major ducts from the parotid and mandibular glands transport saliva to the oral cavity, while the sublingual glands have multiple openings beneath the tongue. In addition, a number of small glands with a number of excretory ducts are present in the mouth.[3] After leaving the mouth, food enters the pharynx and oesophagus. The pharynx is long and narrow. The esophagus is short and covered with stratified squamous epithelium. Beneath the epithelium, a number of submucosal glands are located. Their function is to secrete mucin and bicarbonate, to neutralize luminal acid and protect the epithelium[4].

### 2. 2 The stomach

The stomach of the pigs consists of a simple compartment that is divided into 4 functionally and structurally different regions. The pars oesophagea is a non-glandular extension of the esophagus into the proper stomach. Ulceration – ulcerous autodigestion of the cutaneous mucosa – of the pars esophagea is a common phenomenon in swine production and develops from a complex interaction of dietary particle size, gastric fluidity, dietary carbohydrate content, presence of gastric organisms, and environmental stress factors.

Next to the pars oesophagea is the glandular cardia, which in contrast to most other species is very large and occupies approximately one third of the stomach luminal surface. The fundic, or proper gastric, region is located between the cardiac and pyloric region. All three contain secretory glands located in so-called ‘ gastric pits’. Structurally, they are similar, but they contain different cell types. The major surface of the stomach and lining of the pits are covered with surface mucous cells, that produce thick, tenacious mucus to protect the epithelium against injure from acid and grinding activity.

The gastric pits of the fundic mucosa contain HCl-producing parietal cells that are clustered in the neck of the gland. Distributed between these cells are mucous neck cells that produce thin mucus and proteases. As the only cells of the stomach lining, mucous neck cells divide and migrate either down into the gland or up into the pits and differentiate into any of the mature cell types. Pepsinogen-producing chief cells are located at the base of the fundic glands. In addition, the fundic mucosa also contain endocrine/paracrine somatostatin producing D cells, seretonin producing EC cells, and histamine producing histamine-immunoreactive cells and mast cells (lamina propria)

The cardiac glands have mucous cells that produce mucus, proteases and gastric lipase. The pyloric glands contain gastrin producing G-cells and somatostatin producing D-cells, but the dominating cells are the mucous cells. They do contain mucous neck cells that produce mucus and proteases and zymogen producing chief cells but have no parietal cells. [5]

#### 2. 2. 1 Size and capacity of the stomach

In suckling pigs the pars esophagea, cardic, fundic and pyloric regions represents about 6, 30, 44 and 20 % of the total mucosal area, respectively, while on weight basis the cardia represents only 20 % but the fundic region 56 % of total mucosa weight.

The weight of the stomach represents 0. 5-0. 8 % of body weight in suckling pigs and between 1-1. 3 % in growing pigs. In adult pigs the stomach accounts for approximately 0. 6 % of total body weight. The capacity range from 0. 03 l in the new born to approximately 3. 5 l in slaughter pigs, and 5 l in adults, while under pressure the capacity under increases to 8 and 12 l for slaughter and adult pigs, respectively. A number of studies have shown that the bulk of the diet can influence the subsequent capacity of the stomach. [6]

### 2. 3 The pancreas[7]

The pancreas is located in proximal duodenum. The body of the pancreas separates in the two lobes with the center surrounding the portal vein. A single pancreatic duct leaves the right lobe and enters the duodenum on a minor palpilla 12-20 cm distal to and separate from the bile duct entry, 20-25 cm from the pylorus.[8]

The pancreas is a mixed endocrine and exocrine organ. The exocrine pancreas consists of the acinar cells and the duct system, representing more than 95 % of the pancreas fresh weight. The acinar cells produce and store pancreatic enzymes and inactive zymogens, and when stimulated release them into the duct system for transport to the duodenum. Water, bicarbonate and other electrolytes of pancreatic juice are produced in centroacinar cells and cells of the intercalary and intralobular ducts.

The endocrine part of the pancreas is restricted to the islets of Langerhans. The islet are distributed throughout the acinar exocrine tissue and contain glucagon producing, alpha cells (15-20% of total islet cells), insulin and amylin producing beta cells (65-80%) , somatostatin producing delta cells(3-10%), pancreatic polypeptide producing PP cells (3-5%), and possibly also ghrelin producing epsilon cells (<1%)[9]

### 2. 4 The liver and gallbladder

The porcine liver is divided into 4 principal lobes along with a small quadrate lobe and a caudate process. The lobes, which are the functional units, are surrounded by fine connective tissue. The lobules consist of plates of hepatocytes interdigitated with hepatic sinoids, arranged radially around a central vein. Kupffer cells, which are specialized macrophages, along with endothelial cell line portions of the hepatic sinoids form part of the reticuloendothelial system. Located in the peripheral interlobular connective tissue is the portal triad; the hepatic portal vein, a hepatic artery and an interlobular bile duct, but additionally also lymphatic vessels[10]. Afferent blood from the portal vein and hepatic artery flows centrally in the hepatic siniods. Bile produced by the hepatocytes drains into bile canaliculi formed by hepatocytes and then through ducts of Hering to the interlobular bile ducts in the portal triad. The interlobular bile ducts merge into larger intrahepatic ducts, which become the extrahepatic biliary system. This includes the hepatic bile duct, which divides into a cystic duct connected to the gallbladder, and a common bile duct connecting to the duodenum. The bile duct enters the duodenum on a major palpilla located 2-5 cm from the stomach pylorus.

### 2. 5 The small intestine

The small intestine comprise of the duodenum (4-4. 5%), jejunum (88-91 %) and ileum (4-5 %). The proportion of duodenum in the neonate is similar to that of the adult, whereas differentiation between jejunum and ileum is not clear. Although there are distinctive morphological feature, the duodenum, jejunum and ileum share a lot of common features.

The small intestine consist of 4 major layers; The serosa, the muscularis, the submucosa and the mucosa. The serosa is the outermost layer of the intestinal wall. It has a squamous epithelium forming the mesentery that contains connective tissue, large blood vessels and nerves. The muscular layer contains two types of muscle fibres; an outer layer of longitudal muscles and an inner layer of circular muscles, that are involved in gastrointestinal motility. The submucosa is a layer of connective tissue holding together the large blood and lymphatic vessels and neural complexes. The mucosa consists of 3 sublayers; the muscularis mucosa, the lamina propria and the epithelium. The muscularis mucosa consists of a longitudinal inner muscle and an outer muscle encircling the intestine and produce transient intestinal folds. The lamina propria consists of blood vessels, free lymphocytes and lymph nodes called Peyers patches, and neurons held together by connective tissue. It supports the structure and nourishes the epithelial layer. The epithelial layer consists of a single layer of epithelial cells. They cover the whole luminal surface of the intestine, which is severely folded by the formation of fingerlike projections called villi, and at the base of these Crypts of Lieberkuhn, that are moat-like invaginations.

There are 3 types of epithelial cells on the villus surface: absorptive cells, goblet cells and enteroendocrine cells[11]. They all originate from stem cells located near the base of the crypts. The entocytes migrate from the base to the tip of the villi and during migration, the enterocytes maturate. The digestive function (enzyme activity) begins as the enterocytes migrates over the basal third of the villi. The absorptive function starts to develop as they reach the upper to midlevel and continues to increase until they reach the top of the villi, where they are shed into the lumen. Hence, enterocytes at the surface of the villi are continuously renewed. Goblet cells are secreting viscous mucus, and are interspersed among the enterocytes. Goblet cells increase in number from the proximal jejunum to the distal ileum.

The formation of villi increases the mucosal surface by 10-14 fold compared to a flat surface of equal size. Furthermore, the cell-surface of the enterocytes facing the lumen has an apical membrane forming microvilli (brush-border) that further enhances absorptive surface 14-40 fold. The microvilli have important digestive enzymes and other proteins attached. They extent into a jelly-like layer of glycoprotein known as the glycocalyx that covers the apical membrane. The remaining part of the enterocyte plasma membrane is called the basolateral membrane, referring to the base and side of the cell.

The length of villi increases from the duodenum to the mid-jejunum and then decreases again towards the terminal ileum. This reflects the various functions of the different segments of the small intestine.

Crypts also vary in size and composition along the intestine. They are deepest in the proximal small intestine (duodenum and jejunum) and shorter distally in the ileum. Paneth cells are located at adjacent to stem cells at the base of the crypts[12]. Their exact function is unknown but due to the presence of lysozymes and defensins they most likely contribute to maintenance of the gastrointestinal barrier.

While the duodenum is the site where digesta leaving the stomach is mixed with secretions from the intestine, liver and pancreas, the jejunum is the main site of absorption. Brunner glands, which are located in the submucosa on the part above the sphincter of Oddi[13], produce bicarbonate containing alkaline secretion, which protect the duodenum from the acidic content of chyme, provide an alkaline condition for the intestinal enzymes and lubricate the intestinal walls.

#### 2. 5. 1 Size and capacity of the small intestine

At birth the small intestine is about 2 m long and has a capacity of 72 ml. At weaning it has more than tripled its length (6. 6 m) and has a 9-fold as high capacity (660 ml). The small intestine of fully grown pigs is 16-21 m, weighs 2-2. 5 kg and has a capacity of about 20 l. While the small intestine accounts for approximately 4-5 % during the suckling period, it decreases to 1. 5 % when reaching slaughter weight.

### 2. 6 The large intestine

The pig has a relatively short caecum and a long colon, consisting of an ascending, transverse and descending colon.[14] The caecum is a cylindrical blind sac located at the proximal end of the colon. The cecum, the ascending and transverse colon and the proximal portion of the descending colon are arranged in a series of centrifugal and centripetal coils known as the spiral colon. The caecum and proximal part of the spiral colon has longitudinal muscular bands resulting in a series pouches (haustra)[15]. The rectum is embedded in fat and is dilated to form ampulla recti just before ending at the anus.

The mucosa of the large intestine has no villi, but columnar epithelial cells with microvilli formed into straight tubular crypts. Numerous goblet cells secreting sulphated carbohydrate-protein complex intersperse the columnar cells to lubricate the colon. The rectum has a simple structure with columnar cells and only few goblet cells.

#### 2. 6. 1 Size and capacity of the large intestine

During the suckling period the large intestine is small; From a weight of 10 g and a length of 0. 8 m and with a capacity of 40 ml at birth to 36 g, 1. 2 m and a capacity of 100 ml at 20 d of age. This corresponds approximately to 1. 2 % of body weight. After weaning and during the growing period it grows dramatically (2-2. 5 % of body weight) and increases its weight to 1. 3 kg and length to 5 m at 100 kg with a capacity of approximately 10 l. Adult pigs have a large intestine weighing about 2. 8 kg, a length of 7. 5 m and a capacity of 25 l.

## 3. Function of the digestive organs 3. 1 Salivary secretion (HNL)

Saliva contains a mixture of water (99 %), inorganic salts, mucins, a-amylase. In addition, to serve some protection against diseases, it also contains lysozyme, which breaks down the polysaccharide walls of many kinds of bacteria and immunoglobulin A, which play a critical role in mucosal immunity. Saliva moistens the food, lubricates the esophagus, and initiates the digestion of starch. However, the activity of salivary a-amylase is low, and although secreted in the oral cavity, starch digestion is not believed to be of quantitative importance here, as the time spent in the mouth is too short. Some digestion may on the other hand take place in the proximal part of the stomach prior to acidification with gastric juice. [16] The volume and duration of salivary secretion varies in response to external cognitive or sensory stimuli (cephalic stimulation) and physical and/or chemical stimulation in the mouth. Volume and total activity increases with increased feeding level. However as the ratio of total salivary amylase to total pancreatic amylase is only about 1: 250, 000 in the postprandial phase[17] (0-5 h after feeding), salivary a-amylase may be considered insignificant from a quantitative point of view.

### 3. 2 Gastric secretion (MSH)

Gastric juice is a clear and slightly viscous fluid. The major constituents in gastric juice are shown in Table 1.

#### Triglyceride digestion

HCl is secreted by the parietal cells. However, HCl is not produced within the parietal cell because it would destroy the cell. Both H+ and Cl- are independently transported from the parietal cell into the stomach lumen. Hydrogen ions are generated from the dissociation of carbonic acid that is produced by the enzyme carbonic anhydrase acting upon CO2 and H2O. H+ is then transported to the stomach lumen though a proton pump (H+/K+-ATPase). As hydrogen ions are secreted bicarbonate anions accumulate in the cell. To counterbalance this accumulation HCO3- is exchanged for Cl- at the basolateral membrane. The K+ cations that accumulate within the cells are released back into the lumen in combination with Cl- anions.

HCl plays two important roles in gastric juice. Firstly, it facilitates the protein digestion. HCl denaturates dietary protein, which results in exposure of peptide bonds to proteolytic enzymes. In addition, HCl activates pepsinogen to pepsin and provides a medium of low pH that ensures the optimal activity of the enzyme. Secondly, the low pH provides a non-specific defence mechanism because it inhibits microorganisms from proliferating in the gastric lumen and cause damage to the gastrointestinal tract.

Four types of proteases have been found in the gastric juice of pigs (Table 1). They are all secreted as inactive zymogens (proenzymes that are activated in the lumen) to avoid self-digestion of the cells. The zymogens are activated in the lumen at an acidic pH below 5 or by active pepsin A. Pepsin A is the predominant gastric protease in adult pigs followed by gastricsin. They have strong proteolytic activity at pH 2-3. Pepsin digests approximately 10-15% of dietary protein before it is inactivated in the small intestine[18]. In suckling piglets, chymosin is the predominant protease. It has potent milk clotting activity at pH around 6. Milk clotting is important in suckling animals: it prolongs the passage time of milk along the gastrointestinal tract and enables the thorough digestion and absorption of milk nutrients.

Apart from pepsinogen, the chief cells of the cardiac region of the pig stomach also secrete minor amounts of gastric lipase. This enzyme hydrolyses medium- and long-chain triglycerides and plays a role in the hydrolysis of triglycerides in the stomach of the young pig.

A layer of protecting mucus covers the mucosal surface of the stomach. This layer protects the stomach epithelium from the acid conditions and grinding activity present in the lumen. Mucin secreted by the mucous neck cells of the gastric glands constitutes a major component of the viscous mucus layer.

#### 3. 2. 1 Regulation of gastric secretion

Gastric acid secretion is regulated by gastrin, histamine, and acetylcholine that stimulates while somatostatin inhibits acid secretion.

Gastrin is produced by G cells in the antral mucosa. The production and release of gastrin is stimulated by food compounds mainly small peptides and amino acids and by nervous reflexes activated by gastric distension when food enters the stomach. Gastrin is secreted into the blood stream and acts on the parietal cells via a G receptor. Histamine is an amplifying substance in acid secretion. Histamine is produced by local mast cells and enterochromaffin-like cells and acts on parietal cells in a paracrine fashion. Acetylcholine is a neural transmitter produced by cholinergic neuraon. Acetylcholine is released as response to activation of stretch receptors[19]. The secretion of hydrochloric acid is most efficient when all three regulators are present. Gastric acid secretion is controlled by a feed back mechanism. When pH is 3 or below[20] acid secretion diminishes and gastrin release is blocked. The acidity prevents amines from diffusing into G cells and activate hormone secretion. Furthermore, acid in the lumen causes D cells to release somatostatin. Somatostatin inhibits the parietal cells from secreting acid and G cells from releasing gastrin.

The regulatory mechanisms that control pepsinogen secretion are much less researched but it is generally believed that the pepsinogen secretion is under same regulatory influences as acid secretion.

The gastric secretory activity can be divided into three phases: cephalic, gastric, and intestinal. The anticipation of food stimulates gastric acid secretion. This is controlled by the central nervous system and is called the cephalic phase. The cephalic phase lasts for minutes and prepares the stomach for the entry of food. The gastric phase begins when food enters the stomach. It lasts for hours and accounts for two thirds of the gastric secretions. During the gastric phase acid and pepsinogen secretion is increased. When digesta enters the duodenum the intestinal phase initiates. This phase functions to decrease gastric motility and to reduce the secretion of gastric acid and pepsinogen. The intestinal phase lasts for hours.

### 3. 3 Pancreatic exocrine secretion (MSH)

The primary function of the exocrine pancreas is 1) to provide digestive enzymes for the digestion of the major nutrients and 2) to neutralize the acidic chyme entering the duodenum from the stomach to allow the pancreatic enzymes to function. The pancreatic juice is a clear, colourless liquid that contains salts, bicarbonate, and enzymes. The acini, the functional part of the exocrine pancreas, are composed of acinar cells, that synthesize and secrete the digestive enzymes and ductal cells where fluids and electrolytes originate from.

The main regulatory pathways that control exocrine pancreatic secretion are the hormones secretin and cholesystokinin (CCK) and nervous stimulation.

Acinar, centroacinar, and duct cells have receptors for secretin, CCK, and acetylcholine. When these binding sites are occupied the cells are stimulated to secrete, however, maximal secretion is observed when all receptors are occupied. Secretin is secreted by the endocrine S cells in the mucosa of the proximal small intestine. Secretin is released in response to acid or fatty acids in the duodenal lumen and it stimulates release of bicarbonate by pancreatic duct cells. CCK is released into the blood stream in response to the presence of animo acids, peptides, and fatty acids in the duodenal lumen. CCK is secreted by I cells in the proximal small intestine and it stimulates the secretion of digestive enzymes by the acinar cells. Acetylcholine, released by nerve endings near the pancreatic cells, stimulates secretion. The neurons are stimulated to release acetylcholine by impulses from the enteric nerve system or through the vagus nerve. The sight and smell of food induces vagal responses leading to pancreatic secretion[21]. This is the cephalic phase of pancreatic secretion analogous to the cephalic phase of gastric secretion described previously. Distension of the stomach also causes a vagovagal reflex stimulating pancreatic secretion, which is the gastric phase of pancreatic secretion. When digesta enters the duodenum it evokes a large increase in the rate of pancreatic secretion and the intestinal phase involves both endocrine as well as neuronal stimuli. The distention of the duodenum produces enteric nerve impulses that lead to the release of acetylcholine. The endocrine (hormonal) part of the intestinal phase occurs in response to the chemical stimulation, digestion products of protein and fat stimulates the release of CCK and the low pH of the digesta stimulates the release of secretin.

The exocrine pancreatic secretion is controlled by a feed back mechanism. Diversion of pancreatic juice from the duodenum increases pancreatic secretion. It has been suggested that trypsin is the main component in this feed back regulation as reintroduction of pancreatic juice or infusion of trypsin but not amylase into the duodenum markedly decreased pancreatic secretion. Furthermore ingestion of raw soybeans containing trypsin inhibitor increases pancreatic secretion. There is strong evidence that this feed back regulation is linked with the release of CCK. Enterostatin, a pentapeptide released from procolipase when it is activated by trypsin in the duodenal lumen, may play a role in the feed back mechanism as well. Intraduodenal infusion of enterostatin hs been shown to inhibit pancreatic enzyme secretion.

#### 3. 3. 1 a-amylase

Pancreatic Î±-amylase hydrolyses starch (from plant sources) and glycogen (from animal sources). Starch is composed of amylose, a linear polymer of glucose that is linked by Î±-1, 4 glycosidic bonds and amylopectin, a branched polymer of glucose, that contains both Î±-1, 4 glycosidic bonds and Î±-1, 6 glycosidic bonds. Î±-amylase cleaves the interior Î±-1, 4 glycosidic bonds of starch. During the lifetime of the enzyme-substrate complex amylase hydrolyzes starch by multiple attacks through cleavage of several bonds. The major products of starch hydrolysis are maltose, isomaltose, maltotriose, sugars composed of two or three glucose units, and Î±-limit dextrins, polysaccharides of 5 to 10 glucose residues containing both Î±-1, 4 and Î±-1, 6 glycosidic bonds.

#### 3. 3. 2 Lipases

Pancreatic juice contains three lipolytic enzymes: lipase, phospholipase A2, and carboxyl ester hydrolase, and a protein cofactor, colipase. Lipase is secreted as a fully active enzyme and is the most important enzyme in the digestion of fat. Lipase hydrolyses triglycerides the most abundant lipid in the diet and the products are free fatty acids and monoglycerides. Lipase is strongly inhibited by bile salts in the duodenum and the protein cofactor colipase is the only agent known to counteract this inhibition. Colipase is secreted as a zymogen, procolipase, which requires cleavage by trypsin to become active. Phospholipase A2 splits fatty acids from phospholipids. It is secreted as an inactive zymogen that requires activation by trypsin. Carboxyl ester hydrolase, also known as carboxyl ester lipase and cholesterol ester hydrolase, has an unusually broad substrate specificity, it hydrolyses mono-, di-, and triglycerides, cholesterol and retinol esters and lysophosphatidylglycerols. However, the main physiological function probably is to hydrolyse retinol and cholesterol esters.

#### 3. 3. 3 proteases

The major proteolytic enzymes secreted by the exocrine pancreas are listed in Table 1. All proteolytic enzymes are secreted as inactive zymogens to protect the gland from autodigestion.

The activation of the proteolytic enzymes is initiated by the activation of trypsin by enterokinase, an intestinal brush-border enzyme. Trypsin then activates all other zymogens as well as trypsinogen. Trypsin is an endopeptidase meaning that it breaks proteins at internal points along the amino acid chain, it specifically cleaves peptide bonds on the carboxyl side of basic amino acids (lysine and arginine). The catalytic activity of chymotrypsin is directed towards peptide bonds involving the carboxyl groups of tyrosine, tryptophan, phenylalanine and leucine. Elastase cleaves on the carboxyl side of aliphatic amino acids (alanine, leucine, isoleucine, valine, and glycine). The carboxypeptidases are zinc-containing metalloenzymes. They are exopeptidases meaning that they remove a single amino acid from the carboxyl-terminal end of proteins and peptides.

#### 3. 3. 4 Pancreatic secretion and dietary composition

The enzymatic composition of the pancreatic juice has been shown to be dependent on the dietary composition.

### 3. 4 Bile secretion (HNL)

The bile has pH of 7. 4-7. 9 and contains bile salts, phospholipids, cholesterol (summing up to a total lipid content of 0. 6-0. 7 %), sodium, potassium, chloride, bicarbonate, mucus and bile pigments, of which the latter are endogenous waste products. Bilirubin is a major end product of red blood cell turnover produced by Kupffer cells and transported to hepatocytes for conjugation. The conjugated bilirubin is secreted in the bile responsible for its green/yellow colour. In the intestine conjugated bilirubin is converted by the microflora to urobilinogen, then to urobilin and stercobilin[22] and finally excreted by defaecation, giving faeces its characteristic brown colour. Some urobilinogen is reabsorbed and excreted by the kidney as urobilin, which is responsible for the yellow colour of urine.

Both bile acids and phospholipids play an important role in digestive function, and the molar ratio of total phospholipid to total bile salts is 1: 10. 1[23]. Bile salts are conjugated bile acids, and their function is to aid emulsification and absorption of lipids. The bile acids in porcine bile are mainly conjugated with glycine but also some taurine (6. 5 %). Chenodeoxycholic acid (CDCA), found in the form of 31. 3 molar % glyco-CDCA and 3% taurine-CDCA is de novo synthesized from cholesterol by the hepatocytes. Hyocholic acid (HCA) in the form of 12. 6 % glyco-HCA is produced by hydroxylation of CDCA. Reduction of HCA by the microflora of the intestine leads to formation of hyodeoxycholic acid (HDCA), which in bile is found as 48. 2 % glyco-HDCA and 3. 5 % tauro-HDCA . In contrast to humans, pig bile contains very little cholic acid(CA), found as glyco-CA (1. 3 %). When excreted to the intestine conjugated bile acids are deconjugated and converted by the microflora in the distal small intestine. A majority of the bile acids are reabsorbed in the distal small intestine and transported to the liver via the portal vein. Along with de novo synthesized bile acids they are reconjugated and again excreted in bile. This phenomenon is termed entero-hepatic circulation, and is a mechanism to cope with the demand of bile acids, which by far exceeds the capacity for production. The phospholipids of porcine bile is entirely in the form of phosphatidyl choline, dominated by the 16: 0-18: 2 diacyl forms (59. 6 %), followed by 16: 0-18: 1 (18. 4 %) and 18: 0-18: 2 (15. 9 %). [24]

The bile secretion from the hepatocytes is constant, but bile is only released to the intestine, when needed for lipid digestion. Hence, when little or no food is present in the duodenum, the Sphincter of Oddi is closed and bile is diverted from the bile duct to the gall bladder, where the bile is concentrated. When food, particularly fat-rich food, enters the duodenum, the Spincter of Oddi is relaxed and the gall bladder contracts by a combination of neural and hormonal factors. Gut endocrine cells are stimulated to release CCK, while neurale receptors located at the Spincter of Oddi in conjuction with the intramural plexus coordinates the bile duct and bladder peristalsis.

In bile duct cannulated pigs, where the Sphincter of Oddi is not controlling bile flow, the total bile flow over 24 hours has previously been measured to be 38 and 46 ml/kg in 60 and 45 kg pigs, respectively. Using re-entrant cannulation of the bile duct, which allow gallbladder storage of bile and regulation of flow by the Sphincter of Oddi, it was found that a traditional European pig diet induced a bile 24-h bile flow of 48 ml/kg, while a semi-synthetic diet based on starch, sucrose, casein, maize oil and cellulose led to a flow of 30 ml/kg. Measurement of bile flow by cannulation of the common bile duct and re-entrant cannulation of the proximal duodenum to reintroduce bile at the same rate of excretion resulted in flows of 35 ml/kg for 43 kg pigs fed a wheat-fish meal-casein diet and 59 ml/kg when a similar diet was supplemented with 40 % wheat bran. Hence, the bile flow is influence by the diet. Increasing fat content of the diet from 2 to 10 % induce a dramatic increase in bile acid secretion along with a moderate increase in phospholipid and cholesterol output. A further increase in fat content to 20 % of the diet does not lead to further increase in bile acid flow, while phospholipid and cholesterol output continue to increase. Lipid composition also influences the bile output. While degree of saturation does not appear to influence the rate of bile acid and phospholipid secretion, the secretion of cholesterol is increased.[25]

### 3. 5 Small intestinal digestion and absorption (MSH)

#### 3. 5. 1 Digestion of carbohydrates

The luminal phase of carbohydrate digestion applies only to starches and the enzyme involved is Î±-amylase secreted from the pancreas. Starch hydrolysis products (maltose, isomaltose, maltotriose, and Î±-limit dextrins) and dietary disaccharides (sucrose and lactose) are digested in the membranous phase by digestive enzymes that are a structural part of the intestinal surface membrane.

Four different oligo