Rabbit gastrointestinal physiology

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The rabbit is an herbivore, or more specifically a folivore, designed to exist on a diet of succulent green vegetation. However, its small size means it has a correspondingly high metabolic rate (which limits its ability to exist on a low energy concentration diet), and makes it a highly sought prey (which needs to be agile and athletic to outrun predators). To cope with these problems the rabbit has evolved a digestive tract radically different to that of the better known herbivores such as the horse (a colon fermenter) and the ruminants (gastric fermenters). The rabbit has a system that: (1) allows a high food (and therefore high energy and protein) intake, (2) separates out the digestible and easily fermentable components of the diet, and (3) rapidly eliminates the slowly fermentable fibrous waste that would otherwise have to be carried around. The system also eliminates the need for having a large absorptive surface area in the large intestine by complete separation of the products of cecal fermentation and the feces, allowing reingestion and absorption of bacteria and their by-products in the small intestine. Given that the system is geared for rapid elimination of fibrous wastes, it is somewhat ironic that the main driving force for the system is the presence of such indigestible fiber. Lack of this fiber is the most common cause of gastrointestinal disturbance in the rabbit. This article aims to review the current understanding of the gastrointestinal physiology of the rabbit, and highlights some areas where breakdown of the normal physiologic processes leads to disease. A schematic diagram of the anatomy of the alimentary tract of the rabbit is provided for reference in Fig. 1, and an overview of the activity of the digestive system is provided as Fig. 2.
Fig. 1. Schematic diagram of the anatomy of the alimentary tract of the rabbit. (From Harcourt-Brown F. Textbook of rabbit medicine. Oxford: Butterworth-Heinemann; 2002, with permission.)
Fig. 2. An overview of the activity of the digestive system of the rabbit. (Adapted from Harcourt-Brown F. Textbook of rabbit medicine. Oxford: Butterworth-Heinemann; 2002, with permission.)
**Oral cavity**

**Ingestion**

Rabbits in the wild selectively eat young succulent shoots. To prehend such shoots they utilize the chisel-like incisors to cut off the short pieces of vegetation [1]. This is enabled by the long diastema, rostrally positioned incisor teeth and cleft upper lip ("hare-lip"). Location of food is by means of sensitive vibrissae on the lips, because ocular position in rabbits prevents visualization of objects directly in front of the mouth. The lips themselves are highly mobile, and in captivity (where the diet is composed primarily of long stem hay, vegetable pieces, and pelleted or other particulate foods) the incisors themselves are largely superfluous. Therefore, captive rabbits can cope well with the removal of the incisor teeth.

**Chewing**

Once ingested, the food material is ground down by the cheek teeth (premolars and molars). The teeth in each arcade are arranged in extremely close proximity to one another, and act as a single occlusal surface rather than as individual teeth. The margins of each tooth, and the ridge running from the labial to the vestibular surface across the occlusal surface of each tooth, are composed of enamel. The "valleys" in between these structures are formed from "softer" dentine [2]. The continual attrition that occurs due to tooth-food and tooth-tooth abrasion maintains this file-like occlusal plane on each arcade, and provides an excellent grinding surface. The movements of the jaws and teeth during mastication have been described and illustrated, as have the coordinated tongue movements during the process [3–6]. Masticatory actions are divided into three types. Type I actions are those involved in the slicing actions of incisor use during prehension. Type II actions constitute the main part of the masticatory cycle, providing the chewing and grinding actions necessary for processing of the food and reduction of long stem herbage to shorter particles. Only one side of the mouth is used for this type of mastication at any one time, and the mandible is always moving towards the midline at the stage when pressure is being applied to the ingesta. Type III actions are those involved in forming a bolus of food ready for swallowing. Up to 120 jaw movements per minute have been reported following ingestion of fresh food [7]. When cecotrophs are ingested type II masticatory actions do not take place and the cecotrophs are swallowed intact [8].

**Salivary secretions**

The rabbit has four major pairs of salivary glands: the parotid, mandibular, sublingual, and zygomatic. Amylase and galactosidase are produced in the saliva, which is produced continuously by the mandibular glands, and in
response to food intake by the others [9]. Lipase and urea (prominent in human and ruminant saliva respectively) are only present in trace amounts in rabbit saliva [9]. Potassium and bicarbonate ions are also important constituents of saliva.

**Esophagus**

The esophagus serves as a transport duct from the oral cavity to the stomach. The structure and function of the rabbit esophagus differs little from that of other nonruminant species, and has little or no effect on digestion. No further description is necessary [9].

**Stomach**

*Gastric anatomy*

The stomach of the rabbit is a thin-walled, pouch-like organ. It comprises 15% of the gastrointestinal tract volume [7]. There is an extremely well-developed cardiac sphincter that precludes true vomiting [10]. The cardiac portion of the stomach is thin walled, nonglandular, and intrinsically immobile [7]. Churning of the food material within the cardia is thought to occur indirectly because of large intestinal movements and locomotory movements of the rabbit rather than because of intrinsic gastric motility. The stomach is normally never empty. In fact, after a 24-hour fast the stomach of an adult rabbit has been shown to still be 50% full, usually with a mass of food material and hair surrounded by fluid [11]. Trichobezoars (“hairballs”), originally thought to be simply due to fur ingestion, are now generally considered to be the result of decreased gastric movement, often because of physical inactivity of the rabbit or secondary to reduced motility of the intestine [4]. The fundus is the major secretory portion of the stomach, and has parietal cells (which secrete hydrochloric acid and intrinsic factor) and peptic cells (which secrete pepsinogen, the precursor of pepsin). The pyloric region has a much thicker muscular wall.

*Preweaning gastric physiology and changes at weaning*

Suckling rabbits have a gastric pH of 5–6.5 [12]. Ingested milk forms a semisolid curd within the stomach, which is gradually passed into the small intestine over the 23.5 hours between feeds. The curd formation is due to the action of a rennin-like enzyme [13]. In other animal species, prolonged gastric retention of this curd would be expected to allow marked proliferation of bacteria. However, rabbits at this age have protection against infection conferred by a substance known as “Stomach oil” or “Milk oil”—an antimicrobial fatty-acid product (octanoic and decanoic acids) of the action of the suckling kit’s digestive enzymes on substances in the mother’s milk [14]. Even if bacteria did grow, rabbit kits also have passive maternal antibody
protection (obtained via the placenta before birth, and in the first milk meal after birth) against infectious organisms [15]. These factors maintain the gastrointestinal system of the preweaning rabbit in an almost sterile state. Rabbit kits fed on milk substitutes or milk of another species fail to develop this antimicrobial substance, and severe bacterial enteritis is common in hand-reared kits. Rabbit kits are entirely dependent on milk until 10 days of age [16]. As the youngsters age, they begin to ingest maternal cecotrophs. Because the cecotrophs remain intact for long periods in the stomach within their mucinous coating, the microbial contents may remain protected from the “stomach oil” long enough to pass through into the intestine and colonize the developing hindgut. By 15 days of age some solid material is being consumed. By day 20 solid materials form the majority of the food intake and cecotrophy has begun, and by day 30 milk intake is minimal and cecotrophy is fully developed. During this same period the production of “stomach oil” diminishes. Gastric pH reduces to the adult level of 1–2, which provides another effective barrier against microbial colonization of the stomach and small intestine. The protection of the growing rabbit against enteric infections during the period of weaning depends on the synchronization of this transfer from one protective mechanism to another. Thus, the majority of intestinal disease cases (e.g., coliform infections, coccidiosis, Mucoid enteropathy syndrome, Rotavirus-related diarrhea) in rabbit colonies occur in this period immediately following weaning.

**Postweaning gastric physiology**

In the adult rabbit, large quantities of water and acid are secreted into the gastric fundic lumen. The adult rabbit’s gastric pH during digestion of food material is maintained between 1 and 2, which destroys most microbial organisms, maintaining an almost sterile stomach and small intestine [8,12]. Passage of food material through the stomach has been estimated to take 3–6 hours [17]. Hydrolysis of proteins begins in the stomach, with the pepsin–HCl complex. An important exception is digestion of the mucin covering of the cecotrophs. The cecotrophs are not macerated by the teeth, and remain intact, protected by their mucinous coat, within the stomach for at least 6–8 hours after ingestion [17]. During this time, the cecal material within the cecotrophs is protected from the adverse gastric pH, and microbial fermentation continues, leading to lactic acid formation within the stomach [11]. The pH in the stomach during the period when cecotrophs are present increases to 3 due to the buffering effects of lactate produced by microbes in the caecotroph [18]. The protected environment within the cecotroph has led to the suggestion that feeding intact cecotrophs obtained from a healthy rabbit may be a useful probiotic adjunct in the treatment of various pathologic conditions of the rabbit hindgut (e.g., antibiotic-related dysenterobiosis, enteritis). There are two main problems with this concept. First, it can prove difficult or even impossible to ensure that the cecotrophs are swallowed with-
out breaking the mucin coat. Second, the intact cecotrophs are too large to pass from the stomach to the small intestine until the mucinous coat has been broken down, at which point the bacteria are exposed to the normal gastric digestive processes, and so the bacteria within the cecotroph may not survive to colonize the intestines after all [17].

**Small intestine**

*Small intestinal anatomy and motility*

Gut motility can be divided into a number of different processes. Segmentation is the process involved in the mixing of intestinal contents by periodic static constriction of the intestinal wall, and is particularly important in the rabbit duodenum. Peristalsis is a different process, and involves a ring of contraction moving gradually along the intestine, usually in an aboral direction. The regulation of peristaltic movement involves a number of gastrointestinal hormones and peptides, including cholecystokinin, somatostatin, vasoactive intestinal peptide and “substance P” [8]. Transit time of material through the rabbit small intestine is fast compared to other herbivore species. Peristaltic contractions occur slowly every 10–15 minutes, and do not alter with the stages of the cecotrophic cycle. Chyme retention times have been estimated as 10 to 20 minutes in the jejunum, and 30 to 60 minutes in the ileum [19]. Small intestinal motility in the rabbit, as in the human, but in contrast to most other animal species, is regulated in part by motilin, a peptide secreted by enterochromaffin cells of the duodenum and jejunum. Motilin stimulates smooth muscle contractions [20]. Its release is stimulated by the presence of fats and inhibited by the presence of carbohydrates within the intestinal content [7]. Macrolide antibiotics incidentally also act as motilin receptor agonists, and so may promote smooth muscle contractions [20]. Motilin activity decreases in the distal small intestine, is absent in the cecum, but reappears in the colon and rectum.

At the distal end of the ileum, dorsal to the large intestine in the left caudal abdominal quadrant, there is a round, muscular ampulla referred to as the sacculus rotundus (Fig. 1) [21,22]. This structure seems to have an immunological function and is only found in lagomorphs [22]. It is one of the most common sites for foreign body obstruction of the rabbit intestine [21,22]. An “ileocecal valve” (actually sited between the ileum and the sacculus rotundus) retards reverse flow of fluid into the ileum, and directs chyme via the sacculus rotundus to the cecum [22].

**Pancreatic and hepatic secretions**

As would be expected in an animal with a constantly active digestive system and low protein and carbohydrate intakes the pancreas of the rabbit is small. It is diffuse, and often difficult to locate within the mesenteric fat located between the colon, stomach, and duodenum. The main pancreatic
duct enters near the end of the duodenum, well away from the entry of the bile duct [17]. Although ligation of the pancreatic duct causes dilation of the pancreatic ductules, pancreatic enzymes still appear in the ileal lumen, suggesting the existence of other, minor pancreatic ducts [23]. Trypsin, chymotrypsin, and carboxypeptidases are produced in the pancreas and released into the intestinal lumen. These work along with intestinal aminopeptidases to complete protein digestion. Lipases of various forms are also produced. The pancreas is an important source of bicarbonate ions that neutralize the acidic chyme entering the small intestine from the stomach.

The hepatic ducts drain from the liver parenchyma to the gallbladder via a common bile duct, and thence to the intestine via a cystic duct, which drains just distal to the pylorus. The rabbit produces around 100–150 mL of bile per kilogram bodyweight daily, independent of secretin stimulation—seven times the rate of production in the dog [22]. Bile acids, such as cholic and chenodeoxycholic acids, are synthesized by the liver, and released into the small intestine where a proportion of these is converted by microbial activity to deoxycholic acid [8]. The bile acids are important as detergents that break down fatty or oily material into small micelles, allowing absorption of fats and fat-soluble vitamins in the distal small intestine. The other functional components of bile are the bile pigments. Biliverdin is produced as a breakdown product of hemoglobin, and in most mammalian species is converted by the action of the biliverdin reductase enzyme to bilirubin, before being secreted in the bile. The activity of biliverdin reductase is low in the rabbit, 60 times lower than in the rat, and 63% of bile pigment in rabbits is found as unconverted biliverdin [8,24].

Small intestinal secretive and absorptive physiology

Small intestinal digestion and absorption in the rabbit are similar to that in other species. Bicarbonate ions are secreted in the duodenum to neutralize the acidity of the chyme passing from the stomach. Most of the digestion of carbohydrates and simple proteins takes place in the duodenum and jejunum and the products of this digestion (monosaccharides, amino acids) are absorbed across the jejunal brush border. This includes digestion and absorption of the cecotroph material such as amino acids, volatile fatty acids, vitamins, and digested microbial organisms. The digestion of cecotroph microbial protein is aided by the addition of lysozyme into the cecotrophs as they pass through the large intestine [25]. Lysis of the microbes within the cecotrophs also releases microbial enzymes, notably amylase, which enhances the rabbit’s own digestive processes. The ileum also plays an important role in regulating and recycling the electrolytes secreted by the stomach and proximal small intestine by reabsorbing bicarbonate ions.
Cecum and appendix: anatomy, microbiology, and biochemistry

The rabbit’s cecum is proportionally the largest of any mammal. It is twice the length of the abdominal cavity and 40–60% of the total volume of the gastrointestinal tract [22]. It is a blind sac that folds into four parts (gyri). The first gyrus passes from the umbilical region cranially and to the right across the abdominal floor. It then flexes caudally and the second gyrus passes back, parallel to the first fold, caudally and to the left across the abdominal floor. The third gyrus then passes cranially along the ventral left flank, and runs again parallel to the other folds across the abdominal floor, this time cranial to the first fold and separated from it by the ascending colon. A long fold (the “spiral valve”) extends in spiral form from the cecocolic ampulla at the junction with the colon, along these first three folds, with 20–30 “turns.” These first three gyri have thin, translucent walls. The “vermiform” appendix forms the final fold. It is a 5-inch blind tube that ends on the left flank dorsal to the first part of the cecum and has thick walls containing lymphoid tissue [21,26,27]. The appendix secretes bicarbonate ions into the cecal lumen, which are thought to act as a buffer for the volatile fatty acids produced by cecal fermentation [8,28]. Rabbits fed diets with low fiber and high fermentable carbohydrate develop an enlarged appendix. This has been used as evidence that increased appendix secretory function is needed to counteract the products of increased carbohydrate fermentation. However, it could also be explained by the increased need for lymphoid tissue due to altered microbial populations in such rabbits [8]. Water, secreted by the appendix and colon, is continually added to the cecal contents from where it is absorbed across the cecal wall. This maintains a soft paste to viscous liquid consistency of the cecal contents [4]. The normal pH of the rabbit cecum varies with the stage of the cecotrophic cycle (see below). However, the mid afternoon pH value (the most acidic period) is generally higher in adult rabbits (5.9–6.8) than in weanlings (5.4–6.3) [29]. The cecum provides an anaerobic fermentation chamber for organisms such as Bacteroides spp., which are found at up to $10^9$/g [12]. In addition to the ingesta, mucopolysaccharides secreted from goblet cells in the mucosa serve as a significant carbohydrate source for cecal fermentation by Bacteroides spp. One report describes a large, metachromatic anaerobic bacterium as the most common organism found in the cecal lumen, at $10^8$ to $10^{10}$/mL [29]. Coliform bacteria are rarely isolated from normal rabbit cecal contents. They are suspected to be present in low numbers, and rapidly multiply if any rise in the cecal pH occurs (eg, due to other gastrointestinal disease, or postmortem) [29]. Therefore, bacteriological culture even of a pure growth of Escherichia coli is not necessarily diagnostic of primary E. coli related diarrhea. Other criteria such as epidemiologic evidence and histopathologic findings should also be considered. Various other organisms are normally found in
the cecum. Bacterial species include *Bifidobacterium* spp., *Endophorus* spp., *Streptococcus* spp., and *Acuformis* spp. in the lumen, *Clostridium*, *Peptococcus*, *Peptostreptococcus*, and *Fusobacterium* species adherent to the mucous membrane, and many unidentified anaerobic species [8,19,30,31]. Many nonpathogenic protozoa are found in the cecal contents, including ciliated protozoa morphologically similar to *Isotricha* of ruminants (10⁷/mL), flagellate protozoa such as *Eutrichomastix* spp., *Enteromonas* spp., and *Retortamonas* spp., and an amoeboid organism, *Entamoeba cuniculi* [29,32]. A rabbit specific yeast, *Saccharomyces* (syn. *Cyniclomyces*) guttulatulus is present at around 10⁶/g and is often seen in fecal smears [30]. Lactobacilli are notably absent from the normal intestinal flora of the rabbit [8]. The use of Lactobacilli as a “probiotic” medication for sick rabbits is common. While *Lactobacillus acidophilus* may well be able to survive the rabbit’s gastric pH, its usefulness is widely debated. In the authors’ experience some in-water Lactobacillus products seem to be beneficial to some animals, but this may be because of effects on fluid and electrolyte intake rather than microbial colonization. The combined microbial flora of the cecum breaks down ammonia, urea, proteins, and enzymes from the small intestine and cellulose (preferentially in that order). These microbes also have the ability to metabolize xylan and pectin [19]. The products of this metabolism are the protein and enzyme structures of the microbes themselves (which are later digested as cecotrophs), and byproducts of microbial fermentation referred to collectively as volatile fatty acids (acetate, formate, propionate, and butyrate acids). These volatile fatty acids (VFAs) are actively absorbed through the cecal and colonic walls and utilized by the rabbit as energy sources, as is the case in ruminants. Rabbits differ from other animals in that the level of butyric acid normally exceeds that of propionic acid. Proportions of VFAs in the cecal contents are 60–70% acetic, 15–20% butyric, and 10–15% propionic acid [17]. Both increasing the fiber level of a diet and fasting increase the proportion of acetic acid within the cecal content [33]. It has been suggested that butyric acid may have an inhibitory effect on peristalsis, and hence, the reduction in relative butyric acid level may be one reason why increased dietary fiber promotes gastrointestinal motility [17]. VFAs in the blood are found in similar proportions to those in the cecal contents, suggesting that they are mostly absorbed unchanged into the blood. However, the presence of lactic acid within the blood even when cecotroph ingestion is prevented suggests some degree of parietal metabolism [17]. Prevention of cecotrophy has little effect on circulating levels of VFAs, but high VFA levels within the cecal lumen and blood have been investigated as trigger factors in the initiation of the cecotrophy cycle [17,34].

Colon and fusus coli: anatomy and regulatory physiology

The colon of the rabbit is divisible into a number of different positional and morphologic parts. The ascending colon is very long, and divided into
five limbs extending forwards and back separated by flexures. The first limb has three taeniae forming three rows of sacculations referred to as “haustrae.” In the second and third limbs the taeniae combine to a single taenia and one row of haustra. The remainder of the ascending colon has no taeniae, and lies coiled in the dorsal part of the abdominal cavity. Fecal pellets can first be distinguished towards the end of the ascending colon. The transverse colon is short, and ends in a muscular thickening known as the “fusus coli,” a structure unique to lagomorphs. The separation of the colon into two anatomical and physiologic parts by the fusus coli has led to a renaming of these as “proximal” and “distal” colon, rather than using the more traditional ascending/transverse/descending nomenclature. The fusus coli is a differential pacemaker for the initiation of peristaltic waves in both proximal and distal colon, and regulates the separation (by contractions of the taeniae/haustra) of fermentable material from indigestible fiber [35]. Researchers have shown correlations between fusus coli activity and several potential regulatory mechanisms, including autonomic nervous influences and circulating levels of prostaglandins, aldosterone, and other substances. Aldosterone levels are highest during hard feces production. However, it is not clear whether this is cause or effect of the fusus coli activity. Prostaglandins have been shown to inhibit the motility of the proximal colon and stimulate the activity in the distal colon, aiding the production of caecotrophs [36]. The involvement of the autonomic nervous system and adrenal glands in the regulation of the fusus may be the reason (rather than immunosuppression) why rabbits are prone to stress related gastrointestinal disease. After the fusus coli, the descending colon and rectum return to simple tubular form with thicker walls.

**Food intake and cecotrophy: temporal relativity**

Cecotrophs are pellets produced at the anus from the partially fermented matter of the cecum rather than from unwanted fiber. Because they are not waste material, they are not, strictly speaking, feces, although the term “soft feces” is used synonymously with “cecotroph.” The process of cecotroph ingestion is erroneously referred to as coprophagy. The cecotrophs are ingested by the rabbit directly from the rectum as a result of a neurologic licking response, and are swallowed whole without being chewed. Cecotrophy is influenced by light, ingestive patterns, and varies between captive and wild rabbits [37,38]. In wild rabbits most of the cecotrophy occurs during daytime when rabbits are within their burrows. This is in contrast to the situation in captive rabbits, where most of the cecotroph ingestion occurs at night although it can occur at any time of the day or night [17]. Feed intake patterns inversely follow cecotroph production—when cecotrophy is taking place, food ingestion ceases. This is not a relationship that can be explained by compensatory gastric fill or behavioral satisfaction, as applying collars
preventing the rabbits from ingesting their cecotrophs) does not alter the cecotroph production or the food ingestion patterns [37].

Hunger is stimulated by a number of factors including a dry mouth and decreased blood levels of metabolites such as glucose, amino acids, and volatile fatty acids [12]. Cecotrophy usually follows about 4 hours after ingestion of food. Caged rabbits on a normal daylight pattern and fed ad libitum show an increase in food intake from 3:00 to 5:00 PM, and food intake remains high until midnight. There is then a decreased food intake until 2:00 AM, followed by a further increase peaking at 0:600 AM and ending at 0800 AM [4,19]. Cecotrophy therefore occurs mainly in the periods between midnight and 02:00 AM, and again at 08:00 AM. When rabbits are fed a restricted feeding regime rather than ad libitum, cecotroph production is related to time of food ingestion and loses its influence from light signals [37]. The degree of cecotroph ingestion is directly related to the fiber content of the ingested foods. Cecotroph ingestion is highest when rabbits are fed on a diet high in nondigestible fiber. Dietary fiber is distributed at a set ratio between hard feces and cecotrophs. Cecotrophs have around 50% of the crude fiber level of the “hard feces.” This is regardless of the level of fiber in the food material. However, if dietary protein is restricted, the protein level in the hard feces drops whilst that in the cecotrophs is conserved [8].

*Cecocolonic motility and cecotroph production*

The key part of the rabbit’s digestive process is the regulation of colonic and cecal motility to allow the separation of intestinal contents into indigestible wastes and fermentable substrates. The process is regulated by motility in the colon and can be broadly subdivided into two “phases”—the “hard feces phase,” and the “soft feces phase” or “cecotroph production.”

*The “hard feces” phase*

Initially the cecum is relatively empty, having just expelled its content in the previous “soft feces phase.” Small intestinal material derived from ingested food passes through the ileocecal valve and sacculus rotundus, and is distributed evenly into the cecum and proximal colon. Little cecal fermentation occurs at this stage, and the cecum contracts, expelling most of its contents into the proximal colon. Water is secreted by the proximal colonic wall, which aids the processes of mixing and separation of the contents. Under the control of the fusus coli, three separate contraction types occur [4]. There is a progressive monophasic peristaltic wave of 5 seconds duration, and a segmental low-frequency 14-second duration contraction [4,39]. These both progress in an aboral direction. The third contraction type is that of the haustrae, which undergo high-frequency contractions of 3-second durations, which repeatedly churn the colonic contents [4]. Particle and fluid flow dynamics during this “churning” process dictate that indigestible fibrous particles of greater than 0.5 mm length accumulate within the
central lumen of the proximal colon [40,41]. Smaller particles are moved to
the periphery where they congregate in the pocket-like haustrae [40,41]. The
central fibrous material passes rapidly distally and is formed into “hard
deces” by the physical compressive actions of the fusus coli. Further, water,
electrolyte, and volatile fatty acid absorption occurs as the pellets pass
through the distal colon, and they are finally expelled as small, dry, hard
fecal pellets. They do not have a mucus covering. Digestible components
and fluid, which have accumulated in the haustrae, are passed by retrograde
peristalsis back up the colon and into the cecum for fermentation [41].

**Cecotroph production—the “soft feces” phase**

Following fermentation, the cecal contents form a soft dark green paste,
which is rich in semidigested food material as well as microbial organisms.
Various mechanisms that might trigger cecotroph production have been
suggested, including increased VFA concentration, eating behavior, and the
presence of food materials in the stomach or small intestine, in addition to
the controlling influences over fusus coli function that have already been dis-
cussed. The contractions that functioned to maintain the separation of fluid
and different-sized particles during the hard feces phase now decrease. Peri-
staltic monophasic contractions increase, occurring every 1.5 seconds [4]. The
cecum contracts and the cecal contents are passed rapidly through the colon.
Colonic transit time during the “soft feces” phase is reportedly 1.5–2.5 times
faster than during the “hard feces” phase [35]. The fusus coli contractions
during cecotroph production are more gentle, and do not expel the fluid
from the pellets. Goblet cells in the fusus secrete mucus. As the cecotroph
pellets pass through the distal colon lysozyme is added, and the pellets are
coated with mucus. Cecotrophs arrive at the anus and are ingested directly
in bunches as a response to a number of factors, including rectal mechano-
receptor stimulation, olfactory stimuli, and blood concentrations of various
metabolites and hormones [4,12].

**Summary**

The rabbit’s gastrointestinal physiology is a complex system that centers
around the separation of digestible and indigestible components of the diet
in the proximal colon. The clinical importance of this system is the need for
a consistent diet high in long particle length (>0.5 mm) indigestible fiber to
maintain the motility of the cecum and colon. Most of the common gastro-
intestinal problems seen in captive rabbits are related to inappropriate
diets (low fiber; high protein; high carbohydrate) and infrequent feeding
of treats to which the rabbit is not accustomed. Many of these problems can
be avoided if captive rabbits are fed a diet consisting primarily of fibrous
vegetation, such as grass, hay, and fibrous weeds. Feeding of fruits, grains,
and carbohydrate or fat-based treats should be avoided. Pelleted feeds,
although convenient, should be kept to a minimum, and where pellets are
used those manufactured by an extrusion process, which retains the long particle length of the indigestible fiber, should be chosen.

References
