

MEGACOLON

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RMS is more commonly observed in older rabbits

Photo: R Parry

Having owned many species of pets over the years I am constantly in awe of my rabbits' relationship with food. I don't believe I have come across another pet as food motivated as they are (it is as though we are kindred spirits!). I often joke with other rabbit owners that my rabbits are just a 'stomach covered in fluff' personality-wise, but the same can be said for them anatomically as well.

The digestive system

There is a reason why descriptions of the digestive system of rabbits and gastrointestinal diseases affecting them make up such a large part of the rabbit veterinary text books. Their digestive system is complex and finely balanced, and makes up a huge part of their anatomy. Despite this wealth of knowledge, very little is known and written about Rabbit Megacolon Syndrome (RMS).

To understand more about this syndrome one has to know a little bit about a normal rabbit's digestive function, so that is where we shall start.

Hind gut fermentation

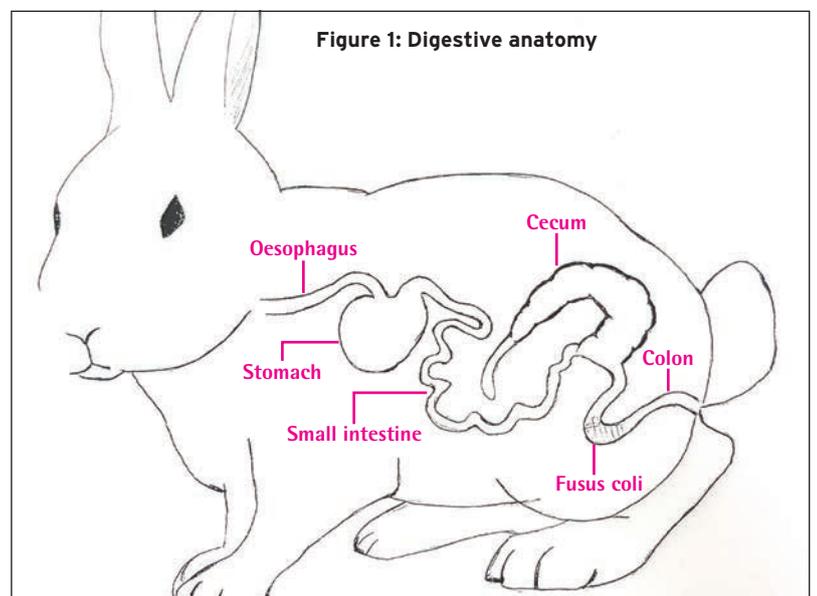
Rabbits are referred to as 'hind gut fermenters', which means the most important digestive activities occur in the caecum, located in the 'hind gut' beyond the stomach (see figure 1). This differs from many species such as dogs, cats and humans, where the bulk of digestion occurs in the stomach (the foregut). In rabbits, food passes down into the stomach which acts mostly as a storage vessel. The stomach makes up 15% of the gastrointestinal tract (GIT) volume and it is here that large volumes of water and stomach acid are added to the food to aid digestion. Food then passes down from the stomach to the small intestine where digestion is continued. Food transits rapidly through the small intestine until it reaches the large intestine which is made up of the colon and the caecum.

It is at this junction between the small intestine, the

caecum and the colon that food is separated into two fractions. Material high in indigestible fibre passes from the small intestine to the colon and out in the form of normal (copious) round poo particles which we know all too well!

Smaller, highly digestible particulate matter moves backwards from the colon into the caecum where it is fermented to form caecotrophs which are then eaten by the rabbit from the rectum. The passage of material through the gut is helped by a wave of contractions of the wall of the intestine known as peristalsis. It is a reduction in this normal movement of the gut wall that veterinarians refer to as 'gut stasis'.

There is a very important and very complex area of the colon which is rich in blood vessels and nerves called the 'fusus coli' (figure 1). This structure is unique to lagomorphs (rabbits, hares and pikas) and it acts as the conductor of the digestive instrument, controlling which particles go



Graphic: N Stapleton

Figure 2: Caecotrophs are covered in mucus, soft and clumped together



Photo: R Lamb

Figure 3: Clinical signs of RMS may include intermittent diarrhoea 'cow pat faeces'



Photo: N Stapleton



Figure 4: Rabbits with RMS are usually considerably underweight

Photo: N Stapleton

forward to be passed out as poo and which go back to the caecum for further digestion.

Ingesta in the caecum is then formed into 'caecotrophs', which differ from normal faeces in that they are covered in mucus, soft and clumped together (figure 2). These are then eaten directly from the rectum as they form an important source of energy, protein, Vitamin B and Vitamin K for the rabbit.

The caecum has ten times the capacity of the stomach and it is filled with a delicate balance of bacteria (both 'good' and 'bad' bacteria) responsible for digesting and making nutrients available to the rabbit. Without this population of bacteria a rabbit would not be able to survive and thrive. When the delicate balance of 'good' bacteria and 'bad' bacteria is upset (referred to as a 'dysbiosis') this can lead to poor digestion, diarrhoea and even death. This dysbiosis is often caused by incorrectly feeding items such as muesli mixes or treats high in carbohydrates and sugars which promote the growth of bad bacteria.

The consumption of caecotrophs varies from rabbit to rabbit depending on the type of diet they are on (high fibre diets promote more caecotrophy) and their health status. For example, sick rabbits may stop eating their caecotrophs and rabbits in pain may find it difficult to eat them.

Despite all that is known about the intricacies of digestive anatomy and function, very little is known about what happens to this system specifically (i.e. at the microscopic level) when things go wrong. For example, diarrhoea could be caused by a vast number of different organisms such as a large number of different viruses and bacteria and other microorganisms such as parasites and fungi, which can act on specific sections

Caecal obstruction due to suspected RMS



Photo: R Saunders

of the gut to cause an abnormality. However, when your pet comes to the veterinarian with diarrhoea it is often given supportive treatment such as fluids, pain relief, syringe feeding etc. It is unusual for us to find out the specific cause of the diarrhoea (viral/bacterial/parasitic) because most rabbits get better on just supportive care without having to investigate further and there may not be specific tests available to find out the underlying cause.

So what happens when we come across a diarrhoea that doesn't get better? What do we do when they get better and then it comes back again? Such is the case with megacolon in rabbits and the answer is: We try and find the cause.

Rabbit megacolon syndrome (RMS)

Rabbit megacolon syndrome refers to a rare disease process observed in rabbits which exhibit chronic, unresponsive, intermittent diarrhoea accompanied by weight loss. The term 'megacolon' refers to the clinical observation that these rabbits often have a very dilated (expanded) colon on investigation. Megacolon is a common problem in cats and although their gastrointestinal anatomy is very different to rabbits the term has been borrowed from them.

The cause of the condition in rabbits is unknown and as yet veterinarians cannot even agree if it is in fact a disease or a symptom of several different diseases, all described under the one umbrella term 'megacolon'. Veterinarians presently disagree if it even exists but having seen two of these cases myself, I am convinced that the syndrome is real and that as yet we lack the means to diagnose and treat it effectively.

Clinical signs associated with the disease vary from rabbit to rabbit but include intermittent diarrhoea 'cow pat faeces' (figure 3) or the formation of faecal pellets of dramatically different sizes. No caecotrophs or normal pellets are produced during these bouts but the faecal output is a combination of high fibre faeces and caecotroph. The rabbits are usually considerably underweight (figure 4) and often fluctuate between a ravenous appetite and complete anorexia. Investigations often show evidence of large amounts of gas in the caecum (caecal tympany) as well as evidence of low proteins and anaemia on blood sampling. None of these signs is specific for one disease and can be seen with many other diseases in rabbits, so as yet there is no one single test that will tell us if your rabbit has megacolon. The condition is only suspected based on clinical signs.

The disease is thought to be genetically based with an increased representation in spotted rabbits such as English breeds. The disease is usually observed in older rabbits and gets progressively worse with time (the bouts of illness become more frequent and severe). Because it is unclear how to treat the condition, many remedies have been tried. Drugs to promote normal peristaltic movement of the gut are often prescribed as well as high fibre diets and intravenous fluid to correct dehydration. Pain relief is important as dilation of the gut is particularly painful. Changes to the diet have been suggested but no one rabbit seems to respond to the same interventions and any improvements are usually temporary, with rabbits having repeated bouts of the clinical signs throughout life. It is my sincere hope that we can determine the cause and cure of this debilitating condition, but until then let us all hope it remains as sporadic as it does mysterious.