KOONAC Goat Farm

Enterotoxaemia in Goats

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1. Introduction

Enterotoxaemia is a potentially lethal poisoning disease of goats, sheep, and cattle, caused by the bacterium *Clostridium perfringens*. It is frequently cited as a common and important goat disease around the world. However, despite this significance, only sparse documentation of caprine enterotoxaemia and its economic significance exists, and not much research has been done that relates to enterotoxaemia in goats.

Our health records confirm that enterotoxaemia is one of the dominate reasons for goats deaths. From 43 goats that died on our farm between October 2006 and April 2019, 11 goats (26%) died from "sudden death", in 11 cases (26%) the reason was “unknown”, and 9 goats (21%) died from enterotoxaemia.

Clostridium perfringens, the bacterium that causes enterotoxaemia, is a parasitic resident of the ruminant digestive tract. It releases toxins. Based on these toxins five main types (strain A to strain E) of *C. perfringens* can be distinguished. However, Type D *C. perfringens* is the principal cause of caprine enterotoxaemia, and enterotoxaemia caused by other *C. perfringens* types are rare.

C. perfringens type D bacteria produce epsilon toxin (ETX), which is considered to be a critical virulence factor. It is the most potent of all *C. perfringens* toxins, and the 3rd most poisonous

bacterial toxin known, behind the neurotoxins of *Clostridium botulinum* and *Clostridium tetani*. LD_{50} of ETX in mice is 70 ng/kg body weight (the equivalent of 1/200'000 g for a 70kg human).

When ETX comes in contact with cells, it damages the cell membrane and makes it permeable for small molecules (<1kDa). It furthermore changes the intercellular levels of potassium, chloride, and sodium. In addition to altering the cell membrane, ETX intoxication also results in cytoskeletal dysfunction. Since these toxic effects of ETX destroy basic cellular functions, ETX leads to unspecific cell dysfunction.

ETX that is released by the *C. perfringens* bacteria in the small intestine causes damages to the intestines, which can be mild to very severe (see Picture 1) Additionally, because ETX enters the blood stream, it also causes systemic damages. Systemic damages are damages to other important organs of the host like liver, kidney, and brain.

Picture 1: Epsilon toxin (ETX) produced by the bacterium *C. perfringens* damages the digestive tract of the affected goat. In mild cases, only the mucosa (the "internal lining" of the colon and the small intestine) is affected. However, in more severe cases some parts of the digestive tract are entirely destroyed. This picture shows a piece of the colon of a goat that was destroyed by ETX and consequently excreted, together with mucosa and many smaller fractions of the colon and the small intestine.

C. perfringens is normally present in low numbers. However, under favourable conditions the population can rapidly grow. Duplication within 8 minutes is possible, which allows the bacterium to grow to more than one million times its original population size within less than 3 hours. It is this potential for rapid proliferation of *C. perfringens* together with the associated release of epsilon toxin that makes enterotoxaemia such a dangerous disease for the affected animal.

2. Aetiology of enterotoxaemia

Enterotoxaemia is also called "over-eating disease", because it can be triggered by a sudden increase of carbohydrate-rich feed, for example due to relocation of the animal from a dry paddock to green, lush pasture, or through accidental access to the feed storage shed. Because the rumen flora is not adjusted to the sudden change of feed, it is unable to digest all the carbo-hydrate input. As a result, the amount of carbo-hydrate (e.g. starch) that bypasses the rumen and is released from the rumen into the small intestines suddenly increases. This can set off a rapid growth of the *C. perfringens* population.

However, the mechanisms that can trigger enterotoxaemia in goats are only partly understood. In particular, feed changes, as outlined above, are not a prerequisite for enterotoxaemia to occur. Mary Smith, in "Goat Medicine" (2nd Edition), reports of an "explosive outbreak of enterotoxemia in a herd of commercial antibody-producing goats that were fed an unchanging diet of hay and concentrate for many months before the outbreak". Our own observations are in agreement with this statement. Of 30 cases of suspected enterotoxaemia, only one case may have been caused by rapid change of feed. All other cases occurred during periods of unchanged feeding regime.

3. Enterotoxaemia incidents at our farm

Different to other ruminants, enterotoxaemia in goats causes severe watery diarrhea, often with mucous, blood and shreds of intestinal mucosa and severe enterocolitis (inflammation of both the small intestine and the colon).

We suspect that 30 incidents of enterotoxaemia occurred on our farm between 2006 and 2019. In 27 cases the goats suffered from severe diarrhea as described above. Furthermore, they were obviously sick and in pain (fever, apathetic, loss of appetite, separating from the herd). Because of these symptoms, these cases were diagnosed as enterotoxaemia and treated accordingly (see Chapter 3.6). Additionally, 3 goats, which also showed typical for enterotoxaemia died without treatment, because they died too fast.

Figure 1 shows the number of enterotoxaemia incidents on our farm for every year together with our average herd size. It can be seen that the number of enterotoxaemia incidents reflects the number of goats we had at this time. Before 2013, when we were farming meat-goats, our herd was bigger than it is now, and we also had more cases of enterotoxaemia then after we had changed to dairy goats and had reduced our herd size.

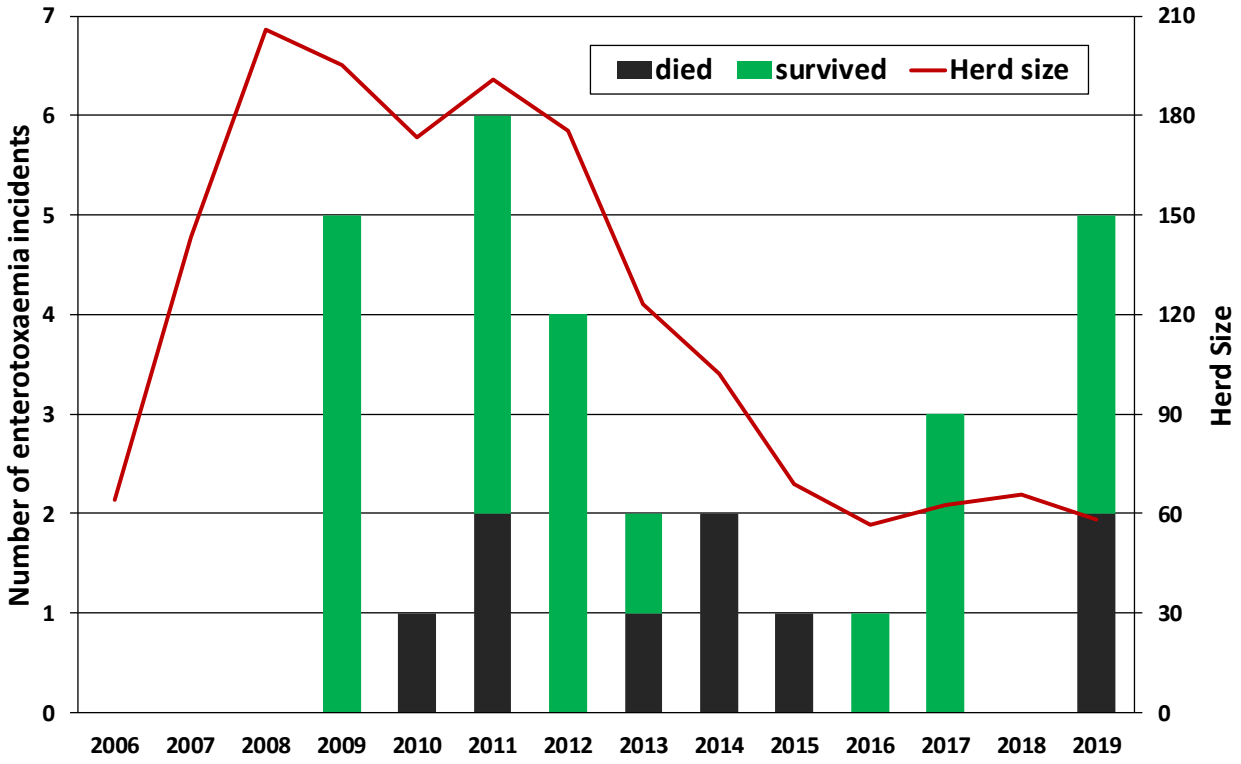

Figure 1: Annual number of enterotoxaemia cases and average herd size.

3.1. Survival rate

According to "Black's Veterinary Dictionary" (15th Edition) enterotoxaemia is "invariably fatal within 36 hours" for goats, and sheep, "seeming perfectly well one day are found dead the next". In "Goat Medicine (by Mary C. Smith and David M. Sherman, 2nd Edition, 2009) enterotoxaemia is characterised as "recognized worldwide as common, frequently fatal disease of goats". And John Matthews, in "Diseases of the Goat" (3rd Edition), lists "death" as clinical sign in both the peracute and acute form of enterotoxaemia.

On our farm, only 30% (9 out of 30) of the suspected incidents of enterotoxaemia were fatal, whereas 70% of the affected goats survived.

Without more data on enterotoxaemia cases available, a reliable prognosis on a reasonable survival rate is not possible. Furthermore, it is not clear to what extend vaccination against *C. perfringens* can protect goats against enterotoxaemia (see Chapter 3.5). However it seems that “frequently fatal” (Mary C. Smith) it probably a more realistic prognosis than “invariably fatal” (Black’s Veterinary Dictionary).

3.2. Age

Most reports on enterotoxaemia conclude that young animals are more affected than older ones. Our data support such an age-related pattern only partly (Figure 2). On the one hand they show that all age groups were affected more or less equally. The occurrence of enterotoxaemia cases in the different age groups is related to size of these age groups.

On the other hand, however, our data seem to indicate that the mortality rate is higher in animals younger than one year than it is in older animals. Of the 11 affected animals younger than one year, 5 (46%) died, whereas mortality rate in older animals was 21%.

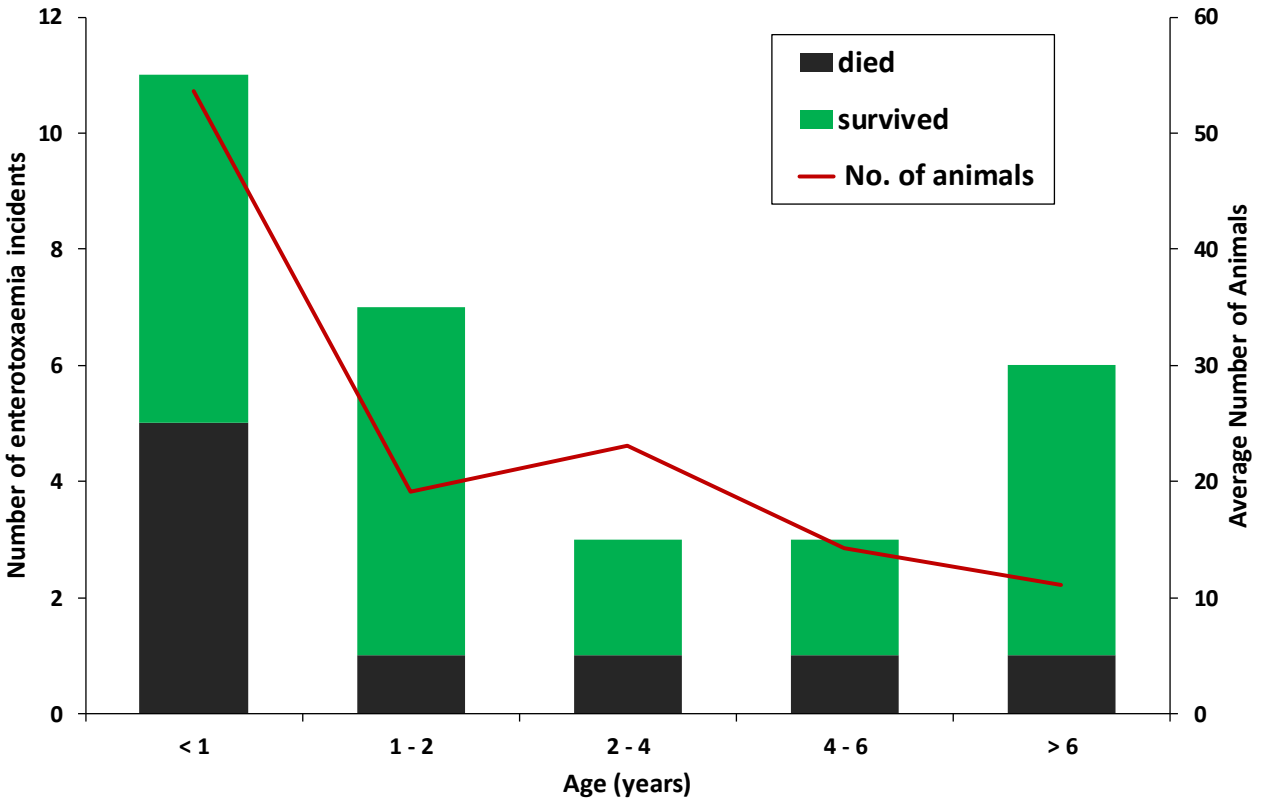

Figure 2: Age of goats that have survived and died from enterotoxaemia on our farm between.

3.3. Seasonality

Enterotoxaemia incidents occurred in all months except June, November and December. Apparently, no simple pattern seems to exist in regards to the seasonal occurrence of enterotoxaemia.

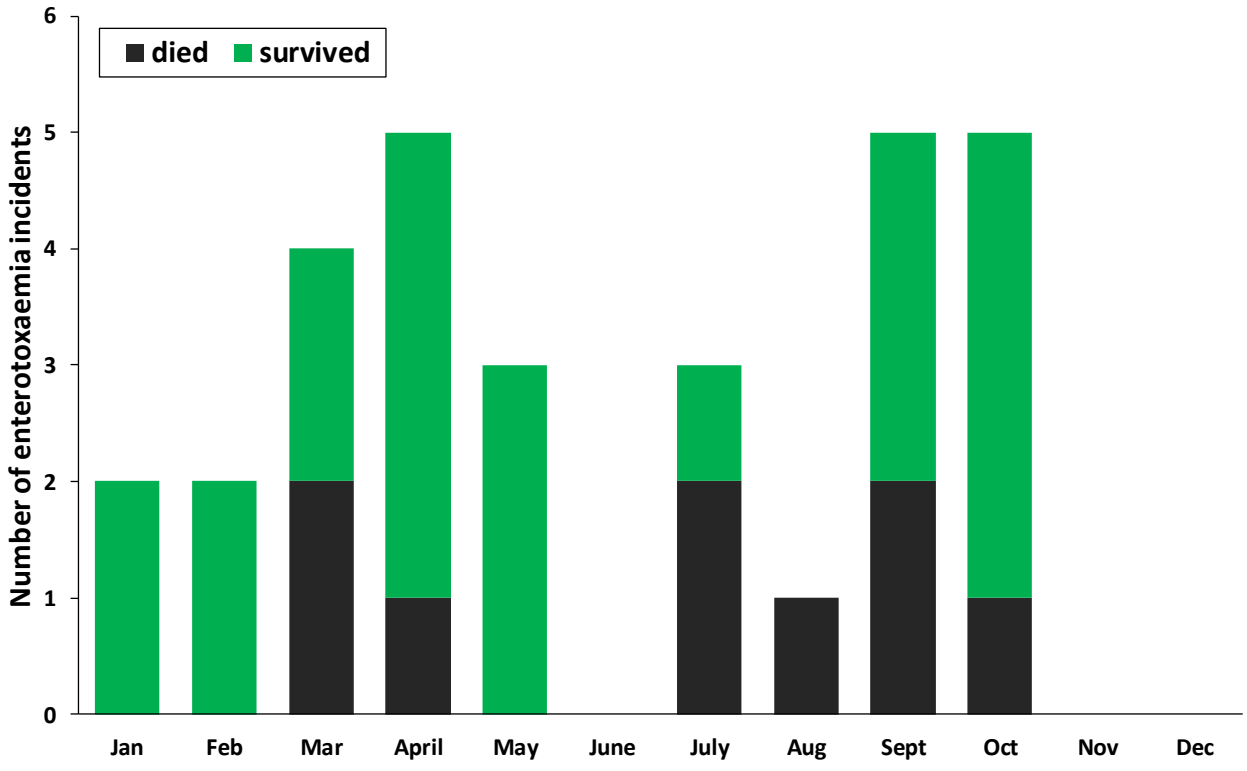

Figure 3: Seasonality of enterotoxaemia incidents between 2009 and 2019.

3.4. Feeding concentrate

As outlined earlier (Chapter 2), a rapid proliferation of *C. perfringens*, resulting in enterotoxaemia, can be triggered when more than the usual amounts of carbohydrates become available in the small intestine. For this reason it seems logic to expect more cases of enterotoxaemia to occur in goats that are fed a concentrate than in goats which do not receive supplementary feeding. However, our data do not support this hypothesis. Only 40% of the enterotoxaemia incidents affected goats that were fed a concentrate (pellets), and mortality of those goats that had been fed a concentrate was 25% (3 out of 12 cases), whereas it was 33% (6 out of 18 cases) in those goats that were not fed a concentrate. Obviously, feeding concentrate is not a prerequisite for enterotoxaemia to occur.

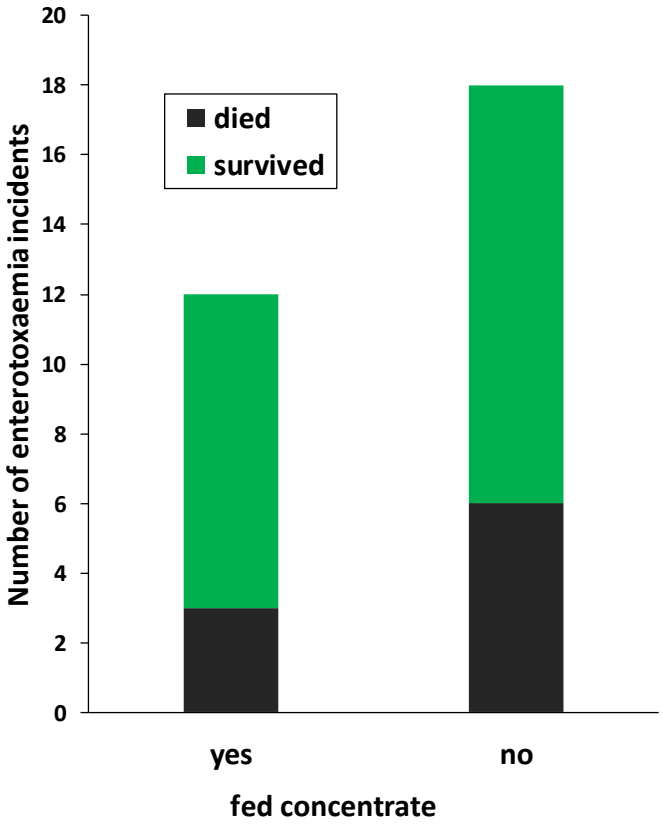

Figure 4: Incidents of enterotoxaemia in goats that were fed a concentrate (left) compared to those that did not receive supplementary feeding.

3.5. Vaccination

All our goats are regularly vaccinated by subcutaneous injection against *C. perfringens* type D. We use Glanvac 3 in 1, which is produced by Zoetis (former Pfizer). It contains at least 5 IU/ml of *Clostridium perfringens* type D toxoid. The goats are vaccinated with 1 ml at 4 - 6 weeks of age, and again about 4 weeks later. Then they are vaccinated with 1.5 ml every 3 months until they are one year old, and then with 2 ml every 6 months for the rest of their life.

The occasional incidents of enterotoxaemia on our farm show that vaccination against Type D *C. perfringens* does not reliably protect goats against this disease. However, we do not know how many (more) cases of enterotoxaemia would have occurred if we would not have vaccinated our goats, and it remains unclear to what extend Glanvac actually protects against enterotoxaemia.

Figure 5 shows that no obvious pattern exists between the occurrence of enterotoxaemia and the period since the last vaccination. Hence, more frequent vaccination is unlikely to improve the protection of the goats against enterotoxaemia.

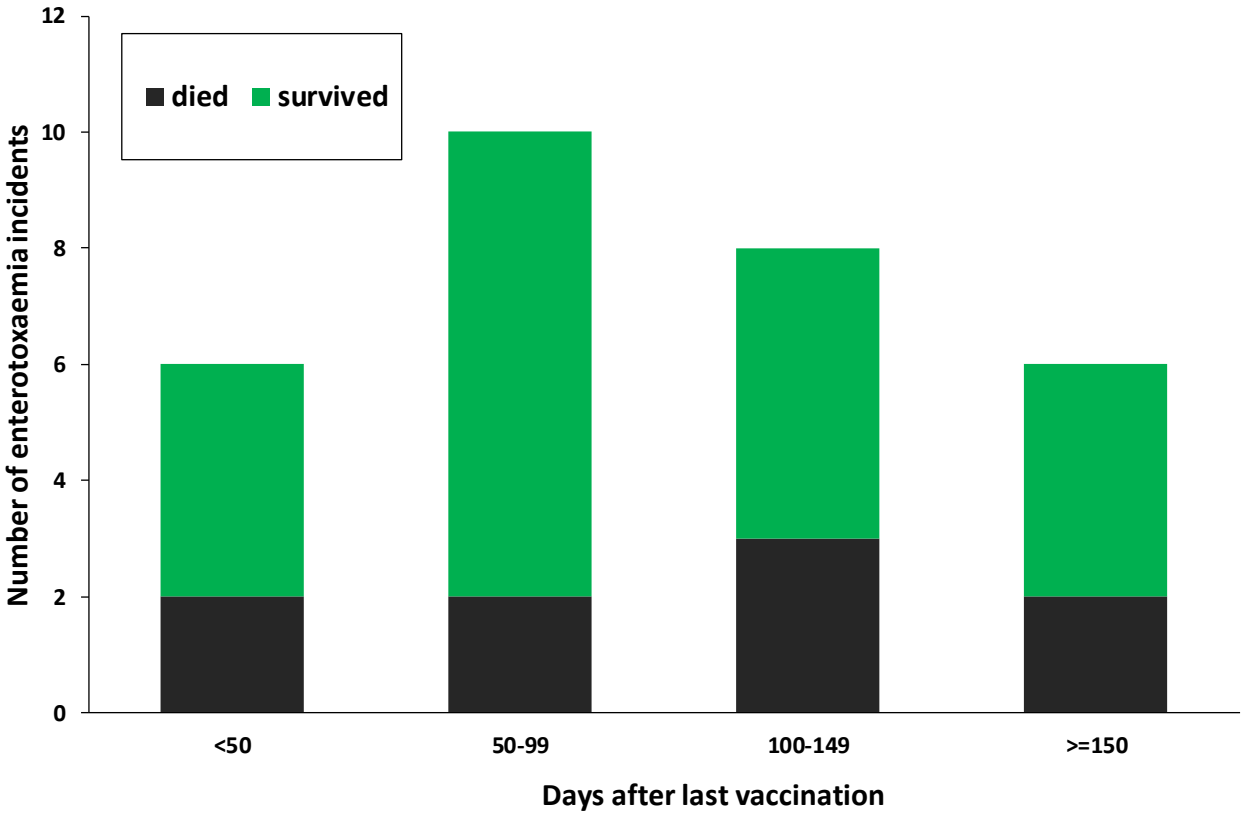

Figure 5: Incidents of enterotoxaemia in relation to the time since the goat had been vaccinated.

Scientific vaccination experiments with goats (e.g. Uzal & Kelly: Protection of goats against experimental enterotoxaemia by vaccination with *Clostridium perfringens* type D epsilon toxoid, The Veterinary Record, 722- 725, June 27, 1998) showed that vaccination induces an immune reaction which protects goats against enterotoxaemia. The protection seems to be weaker, if the vaccination against different diseases is combined than if a monovalent vaccine against *C. perfringens* type D is used. Furthermore, it was suggested that the protection against systemic damages is better that the protection against intestine damages, because the antibodies are mainly present and active in the blood stream, and only to a lesser extend also in the intestine. This could explain why some of our goats survived. They still suffered from a bad, watery diarrhea with mucous, blood and shreds of intestinal mucosa, but the antibodies in the blood prevented ETX to cause fatal systemic damages.

3.6. Treatment

Both, John Matthews (Diseases of the Goat, 3rd edition, 2009), and Mary C. Smith (Goat Medicine, 2nd edition, 2009) recommend *Clostridium perfringens* type D antitoxin as main treatment, and supportive treatment for pain relief and electrolyte balance. Mary C. Smith also mentions antibiotic therapy (administered orally) to reduce bacterial proliferation.

Treatment with antitoxin will not be possible in most practical situations, because *C. perfringens* type D antitoxin is comparatively expensive and usually not available within the short time it would be required.

We normally treat goats with watery diarrhea with an electrolyte solution ("Vytrate", by Jurox Animal Health) to compensate for the loss of minerals and fluid. In suspected cases of enterotoxaemia, or if the goat has not improved after 1 to 2 days despite the electrolyte support, we drench it with "Scourban" (by BOMAC). 20 of the 30 incidents of enterotoxaemia were treated with Scourban, 6 were only treated with electrolyte (Vytrate), but not with Scourban, and 3 cases did not receive any treatment, because the animals had died too quickly. We normally administer 4ml of Scourban per 3 kg bodyweight, which is a somewhat higher dosage than the 15 ml per 25 kg bodyweight recommended by BOMAC. To close the oesophageal groove, which makes sure "Scourban" reaches the abomasum and intestine (i.e. bypass the rumen) we orally administer a small amount of copper sulphate solution before we administer "Scourban".

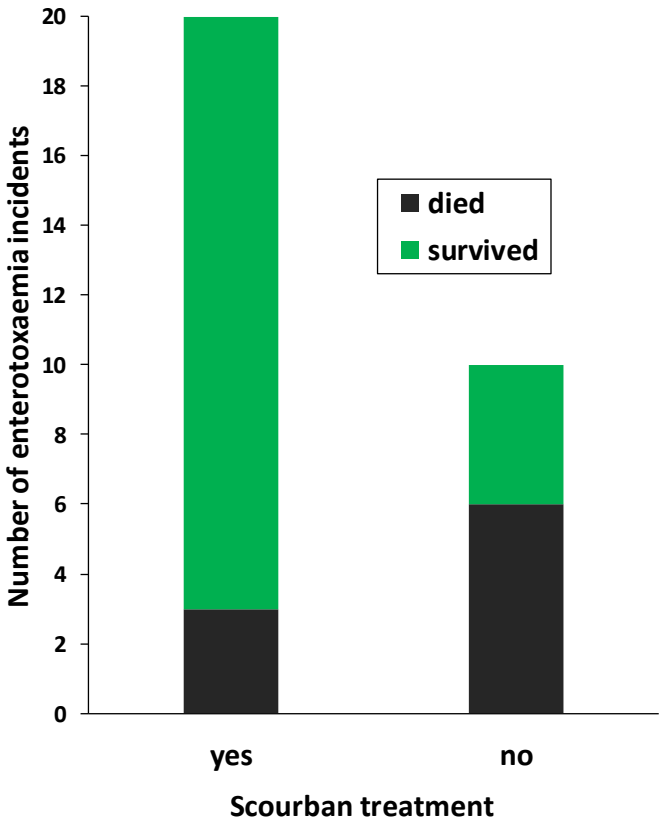

Figure 6: Mortality and survival rate of treatment with and without Scourban

"Scourban" is a drug which is designed to treat bacterial diarrhea in horses, cattle, sheep, goats, dogs and cats. It contains antibiotics (streptomycin and neomycin) and sulphonamides, which exert an antibacterial effect. Furthermore, it contains kaolin and pectin, which absorb bacterial toxins and provide protective coating to inflamed mucosa, hyoscine hydrobromide, which acts as an antispasmodic, and electrolytes (Sodium, Calcium, Magnesium, and Potassium) and glycine, which assist restoration of fluid balance.

Of the 20 goats which were treated with "Scourban", 17 (85%) survived and 3 (15%) died, whereas in the 10 cases where "Scourban" was not administered, 6 (60%) goats died and in 4 cases (40%) the goat survived (Figure 6).

It has to be noted that actually only two of our goats survived enterotoxaemia without "Scourban" treatment, because three out of the four incidents where the goat survived without "Scourban" refer to the same goat. This goat (D146, "Stephanie") suffered from enterotoxaemia five times during her life, but was only treated with "Scourban" twice (at the age of 1.1 years and 4.7 years). She survived enterotoxaemia without "Scourban" at the age of 3.7 years, 7.4 years, and 8.6 years. The other goat that survived enterotoxaemia without "Scourban" treatment was 7.6 years old.

Based on these experiences we think that "Scourban" can substantially improve the chance of the affected animal to survive enterotoxaemia. However, "Scourban" can only save a goat, if the treatment is done in an early stage, before the epsilon toxin has caused too much damage to the vital organs of the goat.

4. Conclusions

We analysed our health records from 2006 to 2019 for patterns that would explain the occurrence of enterotoxaemia in our goat herd. The only pattern that seems to exist is that the survival rate of young animals (< 1 years) is lower than the survival rate of older animals, whereas no correlation could be found between the occurrence of enterotoxaemia and the age of the goat or the season. Furthermore, our data confirm that "overeating" (abrupt change of diet) or feeding concentrate (e.g. grain, pellets) are not a prerequisites for enterotoxaemia to occur.

Because all our goats have been vaccinated, we do not know to what extend vaccination actually protects goats against enterotoxaemia, and how many more would have been affected if we would not have vaccinated. Scientific vaccination experiments with goats have shown that vaccination results in a substantial, albeit not complete protection against enterotoxaemia. Our data do now show a relationship between the occurrence of enterotoxaemia and the period since the last vaccination. Apparently, the reaction of the goat's immune system to vaccination against *C. perfringens* is not simple and not fully understood.

Our data also show that treatment with a drug like "Scourban" that reduces the bacterial growth in the intestine and helps to inactivate bacterial toxins can substantially increase the chance of the affected goat to survive enterotoxaemia.

To gain more knowledge and understanding about caprine enterotoxaemia, more and better data from a broad, coordinated program would be required. In particular, findings from other goat farms should be collected, suspected cases of enterotoxaemia should be confirmed with laboratory tests of samples and post-mortem analyses (if the goat died), and the resulting data should be collated in a central data base.