# Ahidden danger

Tetanus is a devastating disease, but the good news is it's completely preventable. Vet Andrew Robinson from Millcroft Veterinary Group explains why it's so important to ensure your horse is protected

hile everyone's heard of tetanus, not many people know what causes it and how it could affect their horses. Getting to grips with the facts will help you understand why it is vital to protect your horse from contracting this deadly disease, which has a death rate of up to 90%. Although many animals can be affected by tetanus, horses are one of the most sensitive to it, with the possible exception of humans.

### What causes tetanus?

Tetanus is caused by the bacteria *Clostridium tetani*. The environment a bacterium grows and replicates in helps us to differentiate between different families. Some need oxygen to grow and reproduce (aerobes) while others can live in the absence of oxygen (anaerobes). Knowing that *Clostridium tetani* is anaerobic allows us to understand where it is found and how it can cause the disease in horses.

It waits until the environment is right – for example, when there is no oxygen – then it enters the reproductive stage and becomes a viable bacterium that is able to replicate using a process called binary fission. In this process, a single bacterium cell that has reached its maximal growth stage divides into two smaller, identical bacteria cells, which means that the number of *C. tetani* can increase very rapidly.

In anaerobic conditions, when the bacterial cells die they release a potent neurotoxin called tetanospasmin. This causes the clinical signs of tetanus and is one of the most potent toxins currently known – a quantity the size of a pinhead can easily cause disease and death in a horse.

# Our expert



Andrew Robinson BSc (Hons), BVMS, MRCVS is a partner at Millcroft Veterinary Group, a member of XLEquine. He qualified as a vet from Glasgow in 1997 and works mainly on the farm animal and equine side of the practice, but he enjoys working with all the animals that come into Millcroft.



### Sources of contamination

The main places that *C. tetani* lives are in soil, and in the digestive system and faeces of horses, sheep, cattle, dogs, cats, rats, guinea pigs and chickens. Therefore, manure-treated soil may contain large numbers of spores.

Many owners think that the disease is only picked up if their horse injures himself on some rusty barbed wire or a rusty nail. It is true that the bacteria enters the body through wounds if they are dirty, particularly deep puncture wounds, but it can be the dirt on the horse or his environment that is the source of the infection and the nail is only the vehicle by which it can penetrate deep into the tissue. Once in the tissue, the conditions are anaerobic and the bacteria can rapidly multiply.

The environment many horses live in explains why they are very prone to this condition. Puncture wounds on the sole of the foot are probably the most common sites of infection, although it can be acquired via the intestines after eating contaminated soil or droppings, and through gastric or intestinal ulcers. In foals, infection can occur via the umbilicus (navel).

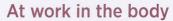
If a small amount of soil or a foreign object causes tissue to die off, the bacteria can begin to multiply at this site. Even routine surgery can be a source of introduction of *C. tetani*, which is why a veterinary surgeon will always check the vaccination status of a horse before an operation.



Soil is one of the



Puncture wounds in the foot are a common site of



Once in the body, the tetanospasmin toxin destroys tissue and decreases the blood supply to the area – a process that further improves the environment for growth and replication of the organism. It enters the bloodstream from the site of infection and binds to the many nerves that are associated with muscle tissue. From the site of the nerve-muscle junction, the toxin travels up the nerves and binds tightly to very specific areas of the spinal cord.

A healthy horse has voluntary and involuntary control over his muscles. An example of a voluntary movement would be thinking about taking a step forward, then moving his limb to take the step. An involuntary movement, for example, would be the body moving itself quickly and automatically as a reaction to touching something too hot.

To move the muscles, whether it is voluntary or involuntary, a nerve signal is sent down the spinal cord that causes the muscles to contract.

These signals do not necessarily result in an all-or-nothing action – often they are a balance between excitatory (positive) and inhibitory (negative) stimuli happening simultaneously. This balance is achieved by cells called interneurons and it is here that the neurotoxin binds, preventing the inhibition of excitatory signals, meaning that only excitatory signals are being sent to the muscle, causing persistent contraction of the muscles.

Once the toxin binds to the interneurons, there is no treatment available that can dislodge it – the effect must slowly wear off. This gives rise to the characteristic clinical signs associated with tetanus and explains why it is also known as lockjaw, because there is a common symptom of tetanus where the jaw muscles become tight and rigid, and the horse is unable to open his mouth. Sometimes tetanus affects only one part of the body, but usually the infection slowly spreads until the entire body becomes stiff and paralysed.



### Signs of tetanus

lameness associated with one limb if that is where the bacteria has entered the body. The third eyelid starts to protrude across the eye, particularly if the horse is startled. The nostrils may also be slightly flared and the lips slightly retracted, giving the appearance of a strange smile. The tail is often held out straight and the horse develops an anxious expression because of facial muscle spasm. Any stimulus such as loud sound, bright light or touch can exacerbate the signs.

The horse will have difficulty moving and eating, may sweat and within 24 hours of the appearance of the initial signs, there will be a generalised spastic activity of the large muscles required for standing. When this happens, the legs become extended in a rigid fashion, so that the horse adopts a rocking horse stance similar to that seen in a pony with laminitis. As the condition develops, the horse will collapse and lie on his side with his legs rigid and extended straight out, and he may suffer convulsions and death from respiratory failure.

The severity of the disease is directly related to the amount of bacteria introduced into the body, which, in turn, determines the quantity of toxin produced.

Puncture wounds on the sole of the foot are probably the most common sites of infection



# Be vigilant

If you are at all concerned that your horse is displaying any of the symptoms described above, you must seek immediate veterinary advice. Prompt treatment is essential to limit the spread of the disease. Supportive therapy must be started straightaway. It is then necessary to identify the possible site of infection, and drain it and flush it to create a clean, aerobic environment that the bacteria cannot replicate in.

regularly and there is a high risk of

To avoid upsetting the horse, sedatives and muscle relaxants can be used, and cotton placed in the ears to reduce noise. Other external stimuli such as light and sound can also be further reduced by stabling or hospitalisation in a dark, quiet stall, with as little handling and disturbance as possible.

If the horse is stabled, attention must be paid to the floor surface. If an affected horse manages to lie down, he can have extreme difficulty getting back up, and if the floor is slippery, it could result in injuries that create more potential entry sites for the bacteria and, at worst, a fracture. A sling may be used to support the horse in the standing position.

In the early stages, if the horse is having difficulty eating and drinking, food and water should be raised to a comfortable height to encourage him to continue feeding. However, if he is unable to eat and drink, intravenous fluid therapy and nasogastric feeding will be used. A catheter may be placed to assist urination and faeces can be evacuated from the rectum manually.

*C. tetani* is quite sensitive to penicillin and large doses will be administered to the horse. In addition to this, a commercially-manufactured antitoxin will be administered intravenously and intramuscularly. This will bind any free toxin that is still circulating in the bloodstream, but cannot neutralise any that is bound to the nervous system. This means that it will take between several days and two weeks to see any improvement in the animal and clinical signs may last a month or more.

## Fatal consequences

Sadly, the condition of the horse may deteriorate to such a degree that the difficult decision may need to be made to euthanase him on welfare grounds, after a discussion between the owner and vet. Sadly, the death rate is 50–90%, but if the horse survives for seven days, the outlook becomes much more promising.

# A simple solution

Given how serious the condition is and how complicated the factors that contribute to it are, it is surprising how few owners take the very simple measure of vaccination to prevent it. An initial course of two intramuscular injections are given four to six weeks apart, followed by the first booster injection at one year and then every second year thereafter.

There may be occasional swelling at the site of the injection or a slight stiffness for a couple of days post injection, which may require some management changes such as raising food and water or even some pain relief. However, the advantages of the vaccination far outweigh the effects of the disease.



Vaccinated pregnant mares should receive a booster 4–6 weeks before foaling, so they are protected should injury occur during the process. Additionally, the foal receives some protection through the mare's colostrum, which can protect him for the first 6–12 weeks of life. To supplement this, foals can be given an injection of tetanus antitoxin soon after birth to provide 3–4 weeks' temporary cover. A regular vaccination programme can then be started when the foal is three to four months of age.

Alongside the vaccination programme, paddocks, stables and stable yards should be kept safe, clean and clear of dangerous items, such as old tractor parts, corrugated iron sheets and building materials that may cause injury. And always pick up poo from the field rather than spreading it – while spreading may kill worms, it will contaminate the land with more bacteria.

If an injury does occur, good first-aid is essential. Wounds should be cleaned as soon as they occur to remove dirt and soil, and veterinary advice sought immediately.