

Ragwort is a commonly encountered weed in the UK. It is potentially deadly to animals especially horses and cattle. The most common form is known as Common Ragwort (*Senecio jacobaea*), but Marsh Ragwort and Oxford Ragwort are also found in certain areas of the British Isles. Common Ragwort has a woody stem with dark green leaves and grows to 30-100cm in height. Its flowers are bright yellow in colour and appear in April/May. It generally dies after it has set seed but can persist and flower for many years. However it is prolific at producing seeds and this is what allows its persistence on grazing land.



All parts of the ragwort plant are poisonous, including the seeds. Even after the plant dies it remains poisonous, so can be a particular problem if it contaminates a batch of hay or haylage. The toxic compound found in ragwort is known as pyrrolizidine alkaloid. This toxin is rapidly absorbed by the intestines and is then transported to the liver where it exerts its effects. In the liver the toxin is metabolised into a toxic compound known as pyrrole. This acts on the liver cells (hepatocytes) and affects them by preventing normal cell division. The hepatocyte becomes enlarged and eventually dies, being replaced by fibrosis instead of new hepatocytes. This damage also tends to be cumulative. The liver can still function normally until approximately two thirds of its reserve has been destroyed. Once this stage is reached, clinical signs begin to appear in a variety of forms, and unfortunately there is nothing that can be done other than treat the horse's symptoms.

Clinical signs of liver failure can manifest themselves in several forms. Many horses will show signs of chronic weight loss (with or without diarrhoea), weakness, general debilitation and jaundice. However, due to the complexity of this organ there are other signs that can appear, which seem completely unrelated to the liver.

One of the functions of the liver is to remove toxins from the bloodstream which have been produced by bacteria in the intestine. One of these toxins is ammonia. It is converted to a product known as urea which is then excreted by the kidneys in the urine. When the liver is compromised this toxin can build up in the bloodstream and begins to have an effect on the nervous system. This appears as aimless walking, standing in a corner with the head pressed against a wall, drowsiness and apparent blindness. The ammonia can also have an effect on the nerve supply to the larynx which leads to the horse having problems breathing.

The liver is also involved in breaking down a toxic by-product from the digestion of grass.

Chlorophyll in all green plants is converted to a product called phylloerythrin by the bacteria in the intestine. Normally this would be removed by the liver but when damaged this product builds up. This product is then activated by sunlight, which is absorbed through the white parts of the skin. It then damages the surrounding tissues and stimulates release of inflammatory mediators. Clinical signs include reddening and inflammation of the skin, ulceration of skin, itchiness and in severe cases the affected parts of skin can completely come off. These signs only appear on the non-pigmented (white) parts of the skin.

Initial diagnosis of ragwort poisoning is based on clinical signs present and the history of the horse e.g. if it has had access to ragwort. This diagnosis of liver damage/failure would then be confirmed by blood tests and a liver biopsy.

Treatment of liver damage and liver failure is aimed at nursing the horse. The idea is that if the damage is not too severe then the given time the liver might be able to regenerate itself to function within normal limits. However, horses that present with liver disease caused by ragwort poisoning tend to have significant damage to their liver by the time clinical signs appear. This means that treatment is **supportive** rather than **curative**. Treatment involves feeding a high calorie diet, with lots of fibre and low protein levels. Sedatives can be given to ameliorate neurological signs and antibiotics can be administered to try to prevent the build up of ammonia levels in the blood by keeping the bacteria which produce it at bay. Ultimately many animals with ragwort poisoning have to be euthanased as the clinical signs have become so severe that the animal's quality of life has deteriorated beyond redemption.

Due to the toxicity of this plant it is classified as an injurious weed and was covered legally by the Injurious Weeds Act 1959. This act states that a landowner can be served an enforcement notice requiring the owner to take action to prevent the spread of injurious weeds if found on their land. However there is now a Ragwort Control Act which gives added protection to horses and other animals. This act was sponsored by the British Horse Society and originated as a Private Members Bill, coming into force in February 2004. It allowed the Secretary of State to put together a code of practice to prevent the spread of ragwort. The code of practice gives advice on identification of the plant, risk assessment of grazing, control methods, environmental considerations and health and safety issues. This act does not seek to eradicate ragwort but control it in areas where it may cause harm to grazing animals. The code of practice can be viewed on the DEFRA website. www.defra.gov.uk