

Ragwort : a growing threat.

Ragwort presents a growing threat to the UK horse population. Dr Derek Knottenbelt of the Liverpool University Veterinary School estimates that around 500 horses and ponies died of liver failure due to ragwort poisoning in 2001. With the increasingly widespread distribution of the plant, he suggests that in 2002 the total number of equine deaths from ragwort poisoning may reach 1000. Even that figure may be an underestimate because the signs are not specific and many cases of ragwort poisoning go undiagnosed.

Common ragwort (*Senecio jacobaea*) is known by several other names, including: St James Wort (because it is in full flower on St James's day - July 25th); Staggerwort (because of its effect on horses); Ben Weed, and Curly Dobbies (in Scotland). It is widespread throughout the country, especially favouring dry overgrazed and neglected land.

Other species of ragwort have a more restricted distribution. All are toxic, as are their smaller relatives the groundsels.

Life cycle

The plant is usually considered to be a biennial (ie it flowers and dies in its second year after growing from seed). During its first year it appears as dense rosettes of ragged green leaves close to the ground. Upward growth, and flowering, takes place in the summer of the second year. The plant grows to between 30cm -100cm tall. It has dense, flat-topped clusters of yellow daisy-like flowers. The flower stems die back after producing large numbers of downy seeds. Each flower head produces an average of 55 seeds ("achenes"). Each plant can produce more than 150,000 seeds, which may remain viable for up to 20 years. Over 70% of the seeds can be expected to germinate.

Under some conditions ragwort can behave as a perennial plant. Most of the plant dies back after flowering, leaving the rootstock to produce new growth the following year.

Toxicity

Ragwort alone probably causes more loss to the livestock industry in the UK than all the other poisonous plants put together.³ Ragwort poisoning takes the form of a slow but certain destruction of the liver. Recovery is impossible once the disease is advanced and signs occur. There is no known treatment that will halt or delay the progression of the disease in the liver.

Ragwort's toxicity is due to substances called pyrrolizidine alkaloids (PAs). Common ragwort contains at least six such active principles including jacobine; jacobine; and jaconine. Related plants have similar toxic alkaloids although they may differ slightly. All parts of the plant are poisonous, including the seeds. Highest concentrations of PAs are found in the flowers and then the leaves. The stems contain lower concentrations of the alkaloids. Although PAs also occur in other plants, common ragwort presents the greatest risk for horses because of its widespread distribution in poorly maintained pasture and in grassland used for hay production.

Pyrrolizidine alkaloids have been found in honey produced by bees foraging in an area of Oregon infested with common ragwort⁴. The pyrrolizidine concentration was estimated to be between 0.3 and 3.0ppm.

Ragwort is probably more toxic in the early stages of growth². In its fresh state, however, the plant appears unpalatable¹ and under lush pasture conditions animals may avoid eating it. Ragwort becomes more palatable following cutting and wilting, and after spraying with herbicides. Under these conditions there is an increased risk of poisoning. Pyrrolizidine alkaloids are not

inactivated by drying, and many cases of toxicosis occur due to ragwort contamination of hay. The toxic dose of dried ragwort is estimated to be 5% of the horse's weight.

Effects on the horse

The PAs are rapidly absorbed from the gastro-intestinal tract. They pass to the liver where they are broken down (dehydrogenated) to produce toxic compounds called pyrroles. Horses and cattle are thought to be more susceptible than sheep and goats to the toxic effects of the PAs. This may be due partly to species differences in the metabolism of the PAs to the toxic pyrroles.

It is thought that the pyrroles disrupt the cell cycle by damaging key genes which control cell division.⁶ Consequently, the hepatocytes are unable to divide and become large cells called megalocytes which are one of the characteristic features of chronic PA poisoning. When the megalocytes die they release toxins which may be taken up by neighbouring cells. The dead cells are replaced by fibrosis, which may itself cause further damage to other cells. Thus the damage to the liver is progressive.

After eating the fresh or dried plant, signs may not be seen for several months. The liver is able to maintain normal function until at least two-thirds of the organ has been destroyed. So the ragwort toxins are able to wreak havoc in the liver without being detected. By the time the horse shows signs of liver failure the damage is so extensive that treatment is not possible. Changes in the liver may take months to develop after the onset of ragwort consumption. Acute poisoning (ie when large amounts of ragwort are eaten in a short time), has been reported but is rare.

Even in cases where small amounts of ragwort have been eaten over a long time, signs appear abruptly and death may occur within a few days. The liver has a major role in carbohydrate, protein and fat metabolism. Any generalised liver disease interferes with most or all of the wide range of metabolic functions. Liver failure, therefore, can produce a variety of clinical signs.

In some cases, loss of condition, reduced appetite and constipation may develop gradually before more obvious signs become apparent. Often the first indication of PA toxicosis is a change in behaviour. Horses may be unable to tolerate the stress of exercise and may show signs of depression, oedema (swelling) of the limbs and loss of appetite after moderate work.

Photosensitive dermatitis (inflammation affecting only the non-pigmented skin) occurs in 25-40% of cases of liver failure in the horse, resulting from the inability of the diseased liver to prevent the accumulation of photosensitizing substances such as phylloerythrin in the peripheral circulation.

As the condition deteriorates, the behavioural abnormalities get worse. They are associated with increased levels of ammonia, aromatic amino acids and volatile fatty acids in the blood, and low blood glucose. Circling or aimless walking is seen in many cases. Jaundice, ascites, subcutaneous oedema and ulceration of the mouth may develop. Secondary impaction of the stomach has been reported. The horse may stand with its head pressed against the wall and become blind. Difficulty breathing associated with collapse of the larynx may occur. In fact, ragwort poisoning is probably the most common cause of bilateral laryngeal paralysis.

The signs can develop so quickly that the cause of death may be wrongly attributed to something else like "heart attack", "stroke" or colic. In some cases the horse may be found dead without warning.

One of the reasons ragwort is so dangerous is that immediate signs of poisoning are not seen. Because the toxicity is cumulative horses can eat ragwort for a long time without showing any sign to alert the owner to the impending death. Once clinical signs start to be seen it is likely that over 75% of the liver has been damaged. There is little chance of recovery when signs are seen.

Ragwort poisoning in pregnant mares has been associated with abortion.⁵ There is a report from Australia of pyrrolizidine alkaloid toxicosis in a two month old foal. The foal died of chronic hepatic damage typical of PA poisoning. During the pregnancy, the mare had been grazing pasture heavily infested with a relative of ragwort, *Senecio madagascariensis*.⁷

Diagnosis

Currently there are no simple tests available to detect ragwort poisoning. There are tests which can detect damage to the liver - in particular looking at liver enzymes in the blood. Liver function can be assessed by looking at bile acids. If the liver function is impaired the levels of bile acids in the blood increase. Neither test is specific for ragwort.

It can also be difficult to confirm that ragwort is the cause of the liver disease because the effects of the toxins are

seen long after the plant has been eaten. There may be no sign of ragwort in the diet of the horse when signs are seen. A diagnosis of ragwort poisoning may have to rely on finding typical microscopic changes in the liver.

Ragwort can be difficult to identify when it is dry in hay. Feed samples can be analysed for PA content. * This is time-consuming, relatively expensive, and the reliability of the result depends upon the accuracy of the sample.

Ragwort blood test

Research at the Liverpool Vet. School has led to the development of a test for early signs of exposure to ragwort.⁸ A pilot study has shown that the test is effective at recognising changes in the blood due to small amounts of pyrrolizidine alkaloids. The test recognises alterations in certain components of the blood cells caused by the toxins. A positive result indicates current or recent access to the toxin in feed.

This simple screening test will make it easier to check whether the food being given is safe or not. Early detection of exposure to ragwort will allow suitable preventative measures to be taken. By stopping access to contaminated food at an early stage, before significant levels of the toxins had been absorbed, it may be possible to prevent irreversible and progressive liver damage.

The development of this test heralds the prospect of reversing the mounting death toll due to ragwort. However, it is not currently available as more funds are required to validate it for general use. Before the test can be available to vets in practice, further work needs to be done to confirm its accuracy and reliability.

A charity, the Ragwort Trust, has been set up to fund the research. Dr Knottenbelt points out that they are looking to raise £80,000 to enable this important work to continue.

Control

Once the clinical signs of liver failure appear in PA toxicosis, there is little hope of recovery, because of the widespread damage and inhibition of regeneration caused by the toxins.

The high mortality seen in all forms of liver failure is evidence of the inadequacies of current treatment methods. In the absence of a effective treatment for ragwort poisoning it is all the more important to prevent horses having access to the plant either at pasture or in

contaminated hay. Preventing ingestion of ragwort is the only way to prevent ragwort poisoning.

Well managed pasture with a dense grass sward discourages germination of the ragwort seeds. Bare patches allow the seeds to germinate. Ploughing, fertilising and controlled grazing strategies should be employed to maintain good quality pasture. With time, this will create a ragwort-free environment for your horse. Some authorities have recommended using sheep to graze heavily infested pastures - on the grounds that sheep are less sensitive to the toxic effects of the plant. Sheep will not eat much ragwort if there is an alternative. However, if they do eat much they are as likely as other species to suffer from its effects.

Preventing ingestion of ragwort is the only way to prevent ragwort poisoning. To control ragwort you need to get rid of the ragwort that is present (before the seeds are shed) and then take steps to prevent it getting re-established.

Control methods.

Pulling.

- ◆ Pulling of ragwort should be done before flowering is complete. It is easiest to do in the spring when the plant is young.
- ◆ Always wear gloves. The toxic alkaloids are easily absorbed through your skin!
- ◆ Remove as much of the root as possible. Digging out the entire plant is the best option
- ◆ Collect all pulled ragwort for burning. Do not leave any lying about because wilted or dried ragwort is much more palatable to horses than the green plant.
- ◆ It is a labour-intensive method which may not succeed in eradicating the plant because remnants of root left behind may re-establish. It will need to be carried out annually on pastures where the plant is well established, because of the remaining seeds in the soil.

Cutting.

This is not generally recommended - because it may actually encourage more vigorous growth in the second year. It may however limit seed production. The plants still need to be collected and burnt.

Spraying

Weedkillers provide an alternative method of control, especially where large areas are affected. A single application cannot be expected to control the problem because of the two year life cycle. Seeds remain in the

soil to germinate in future years. Spraying should be considered annual routine procedure (in autumn) or every other year if done in the spring. Spraying in the autumn is best if you want to cut the pasture for hay.

The rosette stages are effectively killed by spraying. The older plants may have some resistance to spraying.

There are two basic approaches:

- ♦ systemic total weedkiller - applied to the ragwort only (spot treatment) using a knapsack spray, brush or stick.
- ♦ selective weedkiller - such as MCPA or 2,4-D. These are applied more generally using a knapsack spray. One disadvantage of spraying with these compounds is that they will also kill other broad-leaved plants- and so will leave bare patches which are suitable for ragwort to germinate in later.

There is a new selective herbicide available called Barrier H. It is best used as a spot on treatment at the rosette stage. The manufacturers claim it is natural and non-toxic. Animals only have to be excluded for two weeks (but you still have to collect and burn the ragwort!) Advice on herbicide use can be obtained from a BASIS-trained agronomist (see yellow pages).

What can you do when your land is threatened with nearby ragwort.

If agricultural land is threatened by ragwort, you can report the problem to DEFRA. Common ragwort is an injurious weed according to the Weeds Act 1959. The Minister responsible for agriculture can serve notice on landowners to clear ragwort. However, this power is very rarely exercised.

In cases where DEFRA are not concerned, it may be possible to go to court and get an injunction ordering removal of the ragwort and compensation. However, patient negotiation with the owner of the neighbouring land is as likely to be successful.

If roadside verges and waste land are affected, you should contact the local authority.

References.

* ADAS Laboratories, Woodthorne, Wergs Road, Wolverhampton. WV6 8TQ

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Donations to support the development of an early test for ragwort poisoning may be sent to Dr Derek Knottenbelt, University of Liverpool, Leahurst, Neston, Wirral, CH64 7TE, with a covering note to state that the donation is for the Ragwort Research Fund. Cheques should be made payable to "University of Liverpool"

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