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News Release

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Horse sickness shares signs of human brain disorders, study finds

Horses with a rare nerve condition have similar signs of disease as people with conditions such as Alzheimer's, a study has found.

The findings shed new light on the causes of the rare but predominately fatal horse condition and could help to develop new tools for diagnosing the illness.

Scientists say that horses affected by the disease – called equine grass sickness – could also hold clues to human conditions.

Grass sickness attacks nerve cells in horses but the causes of the disease are unknown. It causes gastric upset and muscle tremor and can kill within days. If diagnosed quickly, animals can sometimes be nursed back to health.

Researchers from the University of Edinburgh's Roslin Institute and Royal (Dick) School of Veterinary Studies looked at nerve tissue from six horses that had died from equine grass sickness in a bid to investigate the causes of the condition.

They found that the horse tissue contained proteins that are commonly seen in the brains of people with Alzheimer's disease – such as the build-up of amyloid protein.

In total, 506 different proteins were found to be altered in nerve tissue from horses with grass sickness, compared with animals that had died from other causes.

This knowledge could help to develop tests for detecting the condition in horses, which can be tricky to diagnose.

Around two per cent of horses die from grass sickness each year in the UK. The disease occurs almost exclusively in grass-fed animals, including ponies and donkeys. A similar condition is thought to affect cats, dogs, hares, rabbits, llamas and possibly sheep.

The study is published in the journal *Molecular and Cellular Proteomics*.

Dr Tom Wishart, from the University of Edinburgh's Roslin Institute, who led the study, said: "This is the first study to show similarities between an apparently unrelated neurodegenerative disease of large animals and human neurological conditions. Although the

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causes of these conditions are unlikely to be shared, the findings suggest that similar mechanisms could be involved in the later stages of disease.”

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