

# [Overview of equine diseases](https://assignbuster.com/overview-of-equine-diseases/)

* Pituitary pars intermedia dysfunction (PPID) – Equine cushings disease
* Equine dysautonmia / Equine Grass sickness (EGS)
* Equine gastric ulceration syndrome (EGUS)
* Sweet Itch
* Recurrent uveitis
* Rhabdomyolysis (tying up)

PPID is a disease commonly seen in the equine population with an increase occurrence found in older horses as it is a degenerative condition associated with ageing (McGowan et al, 2013).  Once thought to be a rare condition of geriatric horses, it is a condition that is now found in a large section of horses and ponies 15yrs and over . This is thought to be due to the increase in the number of aged horses and owners seeking veterinary attention and guidance for older horses as they have a better understanding of the disease than before (Sojka-Kritchevsky and Johnson, 2014).

The prevalence of equine gastric ulceration syndrome remains high particularly in competitive horses.  This is predominantly due to some modern management practices that go against the evolution of the horse as a grazing, free moving non ruminant herbivore. These include limited opportunity to free movement, high grain low forage diets, intensive training and stressful environments which all contribute to a poorly buffered and acidic stomach leading to gastric ulcers (Luthersson et al, 2009). There is a greater understanding of preventative measures such as increased turnout, ad lib forage, reduced training intensity and reduce stress but these are not always possible or effective in some horses and combined with the high cost of antiulcer treatment, the frequency of EGUS in horses remains relatively high (Nadeu and Andrews, 2009).

2.

Equine dysautonmia otherwise known as equine grass sickness (EGS) is a polyneuronopathy that affects the central and peripheral nervous system. It is a condition that primarily affects grazing horses and has varying severity of clinical signs but all cases will show signs of neural degradation in the autonomic and enteral nervous system. The acute and subacute cases tend to be fatal but milder chronic cases may recover with intense nursing.  The disease is linked with the bacteria Clostridium botulinum which is found in the soil; however the exact aetiology of the disease is unknown but is thought to be caused by neurotoxicosis (Hunter et al, 1999). The clinical signs usually present as increased heart rate, muscle tremors, patchy sweating, difficulty swallowing, mild colic symptoms, gut ileus, abdominal distention, oesophageal ulceration, drooping eyelids,  rhinitis sicca (dry nose) weight loss or sudden death (Hedderson and Newton, 2004). There is no treatment currently available for EGS so the most effective way to prevent the disease is to minimise the risks. A study in Scotland in the 1970’s which was then matched in by a study conducted by the Animal Health Trust in 1998 identified the major risk factors as; horses grazing 24/7, younger animals aged 2-7yrs, previous occurrence of the disease on the premises, recent changes to pasture or premises with risk decreasing as time passed and absence of hay supplementation (Pirie, 2006). A study by Woods et al suggested there is also climate associated risk factors as cases where identified after cooler, dryer weather and irregular ground frosts.  The findings from previous studies link the condition to a ingested soil borne agent that under certain conditions produces neurotoxins in the horse therefore preventative measures should be taken to reduce the chance of this agent being taken in by the horse. Measures that can be taken include; avoid previous infected paddocks, introduce horses to new paddocks gradually and not during period of high risk e. g after cold, dry period and provide hay supplementation and avoid soil disturbance (Pirie et al, 2014).

Sweet Itch is an allergic skin reaction to predominantly midge ( cullicoides ) bites but reactions can also be caused by mosquitoes and black fly. The condition has been found to affect 5% of the equine population with a high prevalence amongst native breeds. It is the saliva of the biting insects that causes a localised skin reaction and the actual bite can also be painful. The hypersensitivity to the insect varies between horses which will impact on the severity of the clinical signs but the majority of cases will show some degree of pruritus that is concentrated on the mane, neck and tail (Pilsworth and Knottenbelt, 2004). At the affected areas hair loss is usually the first clinical sign to appear but due to the self-inflicted trauma from scratching, over time the area can become sore, bleeding and the skin becomes thickened. Irritability and restlessness can also be present in some horses and in severe cases the distress can lead to weight loss.  There is no cure available for sweet itch and it is a disease that usually gets increasingly worse with time therefore it the best control of the condition is to try and limit the exposure of the horse to midges – the cause of the allergic reaction. These measures include: wearing fly rugs that cover the whole body 24hrs a day, use of fly repellents containing pyrethroid or permethrin based ingredients and stable horses when insects are most active which tends to be dawn and dusk. There are treatments available to control skin irritation but none can cure. These methods include the use of; steroids, antihistamines, anti-itch shampoos, providing essential fatty acids, immunotherapy. A hypersensitivity vaccine is being developed which may be available in the next couple of years will hopefully provide an effective treatment for owners (Chapman, 2019).

3.

Modern equine management practices may increase the susceptibility of horses developing colic as they go against how the horse has evolved. Horses are forage grazing hindgut fermenters that are designed to eat little and often and studies suggest that diet and management has an important influence on the risk of colic (Scantlebury et al, 2015). Horses on a high grain/low forage diet are at greater risk of colic because the intestine has evolved to process a high fibre diet which is essential for gut mobility. The horse has a relatively small stomach which can process effectively small amounts of grain but horses that have large grain or concentrate feeds have an increased risk of colic as some of the concentrate feed may remain unprocessed and pass through into the large intestine causing gastrointestinal upset (). Any changes to diet must be gradually, over 10-14 days, to enable the gut microflora to adjust to the new food that has been introduced. Horses that have a sudden change to their diet are at a great disposition of colic as the sudden change can cause imbalances to the microflora which negatively affects how food is digested. Clean, fresh water must always be available as water is essential in equine digestion as it is consistently secreted into and then reabsorbed by the gastrointestinal tract. Horses with an inadequate water supply are at a higher risk of impaction colic. Other management factors such as limited access to grazing, poor worm control (high worm burden), no routine dental checks  and stress are also predisposing risk factors for colic (Hillyer et al, 2002).

Recurrent airway obstruction (RAO) or otherwise known as severe equine asthma is the name given to nonseptic airway inflammation that is usually induced by an immunological response to organic dust and moulds. The most predisposing factor in the development of the disease is housing and the challenge it causes to the horse’s respiratory tract. There are many contributing factors that are associated with stabling that increases the risk of RAO but they all lead to poor air hygiene as they cause irritantants or allergens to be suspended in the air that are then breathed in and enter the lungs. Forage and in particular dry hay that has been baled with higher than desired moisture content has been associated with RAO due to the high amount if organic particles it produces and in particular the aspergillus fumigatus spores. Horses that are fed from a haynet are more predisposed to RAO compared to being fed off the floor as the nostrils remain in the source of the particles (Ivester et al, 2014). Straw bedding is also linked to the development of RAO due to the high dust content and has been used in studies to induce clinical signs of RAO. Horses kept in poorly ventilated stabling have an increase risk of RAO because respirable particles remain in the air for long periods if there is no or very little through air. A study by McGorum et al (1998) found that respirable particulate and endotoxin levels in the breathing zone where significantly less as pasture than a horse stabled in a low dust environment. Therefore horses that are stabled for any significant time even with low dust management practices are at a higher risk of developing RAO than compared to horses kept at grass.

Pasture associated laminitis the most common form of laminitis seen in the equine population. There are management factors that may predispose horses to this disease. Overweight horses and ponies on unlimited pasture with grass species containing high levels of non-structural carbohydrates are at the greatest risk.  Turning horses out onto paddocks when the grass is actively photosynthesising (lush grass) or when the condition for growth is not optimal (stressed grass) is a common predisposing factor as it is thought that there is more storage carbohydrates in the grass at these times which are thought to be a trigger of laminitis (Harris et al, 2006). Also horses that are not exercised or spend very little time moving around the paddock also have an increased predisposition due to the likelihood of them being or becoming overweight. As study by Alford et al, found a significant higher proportion of acute limits cases happened in the no regular exercise category compared to the control group.

4.

Nutrition has a key role in the development of laminitis and although the exact mechanism is still not clearly determined there is evidence to suggest that a metabolic or digestive disturbance is a contributing factor. If the horse ingests a large amount of poorly digested but highly fermentable food that particularly contains a large amount of starch or fructose (storage forms of carbohydrates) then there is a change to the gut bacterial flora and mucosal permeability (Secombe and Lester, 2012). Studies have suggested that like other mammals, horses do not have the necessary enzymes to digest fructans directly within the small intestine so they therefore pass into the hindgut where they are easily fermented; in a way similar to starch that avoids digestion in the small intestine. This causes some bacteria to die releasing endotoxins which eventually leads to the reduced blood flow to the foot which develops into laminitis (Kronfeld and Harris, 2003).

5. One possible cause of the ponies symptoms is Equine Hyperadrenocorticism / Pituitary pars intermedia dysfunction (PPID) otherwise called equine cushings ‘ s disease.  It is an endocrine disorder of aged horses and ponies that results in the loss of dopaminergic inhibition of the pars intermedia of the pituitary gland which leads to the overproduction of hormones that exert endocrine effects which result in the clinical symptoms of the disease (McGowen et al, 2012). The signs associated with PPID include; delayed coat shedding, hirsutism, weight loss, increased thirst (polydipsia), increased urination (polyuria) and laminitis and some of these symptoms have been described by the owner.  There may be other effects leading to the clinical signs such as insulin dysregulation or equine metabolic syndrome (EMS). This is often a condition linked to the predisposition of PPID however the exact relationship between the two conditions is unknown. EMS increases the likely hood of laminitis due to the excessive hyperinsulinemic response to glucose in the horses’ diet (Krichevsky and Johnson, 2014).

Alongside the presence of clinical signs, the diagnostic test most commonly used is an assay of resting plasma ACTH concentration. A high concentration supports the diagnosis of PPID but it is important that they are interpreted with seasonally adjusted reference ranges in order to gain accurate results. Horses and ponies with unclear results (when they have suggestive clinical signs with normal ACTH results) or in the grey zone (these are clinically normal horses with mildly elevated ACTH levels) should either be resampled during autumn or undergo a TRH stimulation test. Thyrotropin releasing hormone stimulation test is deemed the most precise test for the identification of PPID. The test relies on an extreme pituitary response to the administration of Thyrotropin-releasing hormone (TRH) in horses with PPID when compared to normal horses.

6.

Simple Mendelian diseases are inherited diseases that involve single genes.  The inheritance pattern of single gene diseases is called Mendelian after Gregor Mendel who first observed the different patterns of gene segregation for selected traits in garden peas and was able to determine probabilities of recurrence of a trait for subsequent generations. These diseases are predictable in inheritance as the causative DNA is usually identified in distinct individuals. They can be characterised in groups as dominant, co – dominant or recessive depending on the expression of the mutated allele compared to the normal allele (Finno and Bannasch, 2014). Complex diseases involve the interaction of multiple genes as well as environmental factors. Unlike single gene diseases, complex diseases have a more unpredictable outcome as there is no clear cut pattern of inheritance. Not all horses in the same family will develop the disease but those that do have the right combination of genetic mutation and environmental factors and in some cases the disease will develop regardless of the environmental conditions. This makes it challenging to determine the risk of inheriting or passing on these disorders. Compared to single gene diseases, complex disorders are tough to study and treat because the precise factors that cause most of these disorders have still not be found (Genetics Home Reference, 2019).

Hyperkalaemic periodic paralysis (HYPP) is a Mendelian autosomal dominant genetic disease that is seen in quarter horse breeds.  The disease emerged as a natural occurring genetic mutation that has been passed on through selective breeding (for desirable pronounced musculature) as HYPP can be traced back to a single breeding sire. The most common clinical signs of the disease are muscle twitching and tensing. In mild attacks they remain standing and the recovery can be spontaneous but in severe cases the horse can display weakness by swaying, buckling at the knees, paralysis of hindquarters and involuntary collapse. The symptoms are usually accompanied by sweating, slightly increased heart and respiratory rate and decreased tendon reflexes.  The disease can be indicated by high potassium levels in serum which can help in the diagnostic testing for the disease. The episodes of HYPP are unpredictable and very in severity but can occur; after sleep, rest after exercise, during or after a period of stress, traveling or surgery.  Usually horses that are homozygous are more severely affected than heterozygotes and as the disease is autosomal dominant there is no gender difference in developing HYPP.  The most effective treatment is controlling the potassium contractions in the serum which is can managed through diet and medication using acetazolamide a potassium wasting diuretic and carbonic anhydrase inhibitor (Meyer et al, 1999).

Osteochondritis dissecans (OCD) is a complex developmental disease in horses that affects the bones and cartilage of joints and is a cause of lameness and decreased performance in young athletic horses. The disease causes the cartilage in the joints to form abnormally causing the cartilage and the bone underneath to become irregular and thickened and forms bone and cartilage flaps that either are partially attached to the bone or break off and float in the joint space (Bates et al, 2015). This then causes an inflammatory response in the area which overtime may develop into arthritis.  Studies have found there is a genetic component to the development of the disease but environmental factors such as; nutrition leading to high growth rate, exercise, trauma and hormone imbalance are also key in the disease formation. Alongside lameness, the most common clinical sign is swelling at the joint and it is most commonly seen in the hock, fetlock and stifle joints (Weeren and Olstad, 2015).

References

* Alford P, Geller S, Richrdson B, Slater M, Honnas C, Foreman J, Robinson J, Messer M, Roberts M, et al (2001) A multicenter, matched case-control study of risk factors for equine laminitis. Prev Vet Med. 49pg 209–22.
* Bates, t. , Jacobs, J., Shea, K., Oxford J. (2015) Emerging genetic basis of osteochondritis dissecans. Clin Sports Med . 33(2) 199-220
* Chapman, S.(2019) Get ahead of sweet itch. Equine Health.
* Finno, C., Bannasch, D.(2014) Grayson-Jockey club research foundation: Review article Applied equine genetics. Equine Veterinary Journal . 46(5) pg 538-544
* Genetics Home Reference (2019) What are complex or multifactorial disorders? [online]https://ghr. nlm. nih. gov/primer/mutationsanddisorders/complexdisorders[accessed 24. 05. 19]
* Harris, P., Bailey, S., Elliot, J., Longland, A. (2006) Countermeasures for Pasture-Associated Laminitis in Ponies and Horses. The Journal of Nutrition . 136(7) pg 2114 – 2121
* Hillyer, M., Taylor, F., Proudman, C., Edwards, G., Smith, J., French, N. (2002) Case control study to identify risk factors for simple colonic structures and distention colic in horses. Equine Veterinary Journal . 34(5) pg 455-463
* Hedderson, E., Newton, J.(2004) Prospects for vaccination against equine grass sickness. Equine Veterinary Journal . 36(2) 186-191
* Hunter, L., Miller, J., Poxton, R.(1999) The association of Clostridium botulinum type C with equine grass sickness: a toxicoinfection? Equine Veterinary Journal . 31(6) 492-499
* Kronfeld D, Harris P (2003). Equine grain-associated disorders. Compend Contin Educ Pract Vet . 25pg 974–83.
* Luthersson, N., Nielsen, K., Harris, P., Parkin, T. (2009) Risk factors associated with equine gastric ulceration syndrome (EGUS) IN 201 horses in Denmark. Equine Veterinary Journal . 41(7) pg 625-630
* McGowan, T., Pinchbeck, G., McGowan, C. (2013) Prevelence, risk factors and clinical signs predicitive for equine pituitary pars intermedia dysfunction in aged horses. Equine Veterinary Journal . 45pg 74-79
* Meyer, T., Fedde, M., Cox, J., Ericksom, F.(1999) Hyperkalaemic periodic paralysis in horses: a review. Equine Veterinary Journal. 31(5) pg 362-367
* Nadeau, J., Andrews, F.(2009) Equine gastric ulcer syndrome: the continuing conundrum. Equine Veterinary Journal. 41(7) pg 611-616
* Pilsworth, R., Knottenbelt , D. (2004) Equine Insect Hypersensitivity. Equine Veterinary Education . 16(6) 324-325
* Pirie, R.(2006) Grass Sickness. Clinical Techniques in Equine Practice.
* Pirie, R., Jago, R., Hudson, N.(2014) Equine Grass Sickness. Equine Veterinary Journal . 46pg 545 – 553
* Sercombe, C., Lester, G. (2012) The role of diet in the prevention and management of several equine diseases. Animal Feed Science and Technology . 173pg 86– 101
* Sojka-Kritchevsky J, Johnson, P.(2014) Current status and future directions: Equine pituitary pars intermedia dysfunction and metabolic syndrome. Equine Veterinary Journal . 46pg 99-102
* Weeren, P., Olstad, K. (2015) Pathogenesis of osteochondrosis dissecans: How does this translate to management of the clinical case? Equine Veterinary Education . 28(3) 155-