

Equine Metabolic Syndrome

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Over recent years, a new metabolically related condition has been identified and is now recognised in horses and ponies from as young as 12 years of age and more commonly in horses over 18 years of age. New research has linked an Equine Cushing's-like syndrome, referred to as **Equine Metabolic Syndrome**, or EMS for short to a history of high GI sweet and processed feeds, heavy weight condition, 'good doers', abnormal fat distribution, lack of exercise and recurring laminitis (founder) in aged horses, ponies and minis.

It is now more clearly understood that EMS is related to abnormal glucose metabolism and insulin resistance, rather than the previously held belief that these symptoms were consistent with hypothyroid activity which developed as a horse ages.

How Does Equine Metabolic Syndrome Develop?

Many horse and pony owners are aware of Equine Cushing's Disease (ECD) in aging animals, but EMS often develops in middle aged horses and ponies that have a lifetime history of overweight and heavy condition, especially when grazed predominately on improved pastures, fed on highly processed 'sweet feeds', or a diet high in soluble sugars or non-structural carbohydrates (NSCs) as grain starches, or a base diet of high quality grass hay (especially rye grass-clover hay) to maintain them in show condition for a greater part of the year. Many of these horses do not have a lean or 'strip off' period over winter to readjust their glucose metabolism.

Often by 12-14 years of age, a metabolic change occurs as glucose intolerance and insulin resistance (IR) develops, which then displays the characteristic signs of an even higher risk of obesity and a battle to maintain a moderate condition, even on maintenance rations.

In common with Equine Cushing's Disease, affected animals develop exercise intolerance, a long hair coat, abnormal fat distribution, and a repeated history of laminitis episodes and acute sensitivity to changes in sugar intake from pasture or hard feeds and changes in the glucose or starch content of their diet. They often develop the appearance of long toes, flat heels and recurring bouts of lameness associated with chronic founder, now linked to insulin resistance.

Heavily pregnant mares over 12 years of age are particularly susceptible to the effects of Insulin Resistance. This is because of the suppression of insulin activity on blood glucose in the last 8-10 weeks of pregnancy when the unborn foal has preference on glucose supply through the membranes to increase its body weight by 45% until it is born.

EMS is a term that describes the syndrome related to combined glucose intolerance and insulin resistance, similar in many respects to Type 2 Diabetes with increasing obesity, a sedentary lifestyle and altered glucose and insulin sensitivity that develops in men as they reach 50-60 years of age.

What Causes Insulin Resistance?

An endocrine gland disorder, such as developing IR as the horse ages, can have a direct effect on the onset of ECD and EMS, especially in horses that have a history of continued heavy condition and starch based grain diets.

At this time, research has not linked the risk of pituitary gland dysfunction leading to ECD, or pancreatic gland disorders in the case of EMS, to breed or bloodline, although some Thoroughbred and Warmblood bloodlines, pony breeds and miniatures tend to have a higher incidence because of their 'easy keeper' metabolism or tendency to be overfed relative to their exercise programs.

Equine Cushing's Disease appears to be common in middle to late aged grey ponies. This type of pony is very popular with children and there are many in retirement in the back paddocks of semi-rural areas in the outskirts of cities and towns. Many of these ponies are overweight and have a long shaggy coat which grows rapidly after a clipping. **They are plagued by sore feet, white line disease and recurring laminitis.** ECD was originally classified as a metabolic disorder related to pituitary gland dysfunction (PPID), resulting from a tumour growth in aged horses and responsive to medication with the drug pergolide. However, not all horses that have the signs characteristic of ECD are responsive to pergolide therapy. Many of these horses were found to have abnormal glucose metabolism or an intolerance to glucose due to developing Insulin Resistance, now classified as non-pergolide responsive EMS.

Insulin resistance (IR) is a result of decreased tissue responses to circulating insulin hormone which acts to control blood glucose within a set and safe metabolic range. This causes a decrease in the insulin-mediated glucose uptake in skeletal muscle tissue, fat storage tissues and the liver. Studies have shown that in IR affected horses and ponies, the insulin sensitive tissues are less responsive to glucose when insulin is administered and blood glucose levels are monitored over a 4-6 hour period.

In the most common form of IR, classified as compensated IR, which develops in horses and ponies, serum insulin concentrations in the circulating blood are higher than normal because more insulin is being secreted from the pancreas to compensate for the lower tissue uptake of glucose. The mineral magnesium has a role in normalising tissue uptake of glucose in IR resistant animals.

In some older horses, or following disease where the pancreas is unable to produce enough insulin, resulting in lower circulating insulin levels relative to the concentration of glucose in the blood, the changes are referred to as **non-compensated IR.**

Many horses with advanced ECD associated with the PPID form, become diabetic, because PPID increases circulating cortisone levels as a result of pituitary cancer. It acts to reduce glucose uptake into the normally insulin sensitive tissues, increasing circulating glucose levels in the blood (hyperglycaemia) because the glucose supply to high priority tissues, such as the brain, is increased during times of stress or danger when horses have elevated blood glucose levels to fuel the brain tissue.

Other Causes of Insulin Resistance

The stress of hard training and competition can increase the risk of IR. The administration of cortisone drugs to help reduce joint inflammation and skin reaction associated with the itch can trigger a transient IR and obese animals already 'bordering' on clinical IR, may suffer an IR crisis and develop a secondary laminitis and founder.

Insulin resistance increases naturally during the last 70 days of pregnancy in a mare as more glucose is delivered through the membranes, unaffected by the action of insulin, to provide the energy needed to fuel the very high rate of development where the unborn foal increases its body weight by 45% in the last 2-3 months before foaling.

Obesity and Insulin Resistance

Although IR is a metabolic consequence of obesity in horses and ponies, in older horses gaining weight due to high energy rations and a more sedentary lifestyle during retirement, weight control by dietary management to reduce starch and NSC intake, including limiting pasture grazing when plants are in their early, rapid phase of growth after rain or irrigation, must be instigated before the horse becomes obese and progresses to clinical IR.

However, some IR horses may not be overly fat or obese, but as a consequence of developing IR, they may progress to clinical IR with signs of abnormal fat distribution, such as a 'cresty' neck, fat pads around the tail base (butt), bulging fat deposits above the eyes and increased soft pads of fat behind the shoulders and under the belly. Normally the development of a 'cresty' neck is one of the most characteristic signs of early IR in otherwise reasonably conditioned and healthy animals.

Exercise and Dietary Management

Equine Metabolic Syndrome (EMS), unlike the other similar Cushing's-like symptoms related to a pituitary gland cancer, can be effectively controlled by a combination of limiting grazing and hard feed restriction and daily exercise.

The aim of dietary control is to reduce body fat mass and abnormal fat distribution so as to improve insulin sensitivity, by selecting 'low glycaemic' feeds (GI), combined with other dietary measures and increased, but not excessively hard exercise that would otherwise increase blood glucose responses.

Careful management will also reduce the progression to laminitis and founder in clinically IR horses and overweight ponies. Obviously in PPID horses with developing ECD, pergolide therapy should be used in conjunction with dietary and exercise management to control IR and its clinical symptoms. This will help normalise metabolic function and in most cases, improve the horse's lifestyle for 3-5 years until the severity of PPID becomes non-responsive to pergolide therapy.

1. Restriction of pasture access to 1-2 hours total per day.

Keep pastures short and if necessary purchase a grazing muzzle so that the horse or pony can exercise without consuming too much 'sugary' (non-structural carbohydrate NSC) grass, especially rye grass based pasture in its early dynamic phase of growth in early Spring, or a growth flush following late summer or early autumn rains, which often cause an increase in the risk of laminitis due to excess fructan or NSC carbohydrate intake as a result of unrestricted grazing

2. Ensure a regular exercise program - daily walks for 15-20 minutes without grazing to utilise excess blood glucose.

3. Controlled intake of NSC sugars in hard feeds and hay, especially good quality grass hay which was early growth phase pasture before being cut and cured as hay, as it can be even more dangerous in small amounts as dehydrated pasture in the same way that it was as rapidly growing, high NSC yielding, lush grazing pasture.

Choose low GI or low NSC feeds by avoiding cereal grain based pellets, sweet grain mixes or grain based extruded feeds. Feed

restricted amounts of millrun based equestrian pellets, rice bran (it too contains 20% NSCs) and even copra meal. Feeding Omega balanced vegetable oil blends (oils are very low or zero GI feeds) at a rate of up to 5% by weight of the ration, introduced in a step-wise manner over 10-14 days, will help ensure efficient digestion and reduce blood sugar levels as a part replacement for grain or high GI feeds. Ideally, the daily hard feed and hay should be split into 2-3 meals, with some during the day to avoid a large meal, especially at night, which can lead to a higher glucose peak and compensatory IR. Low GI feed products such as Equi-Jewel® and Rebuild® are also recommended, but it is important to feed only the amounts recommended, preferably split between 2-3 feeds. This is also important when feeding other commercial feeds that are recommended to help avoid laminitis and founder, especially to over-weight horses with classical symptoms of IR.

Suitable roughages include hay which has been soaked in double its volume of warm water for 60 minutes and then taken out to drain and air dry before feeding. As a part or full substitute for oaten chaff, feeding 5% of the total ration weight of dry sugar beet fibre and then soaking it for 10 minutes before feeding to supply low GI, highly digested fibre or 10-15% by weight of lupin or soyabean hulls, will help to minimise the glucose peak after feeding. Remember that sugar beet fibre contains some NSC - so avoid large single feeds, especially in overweight horses and ponies. **Good quality oaten chaff has a higher GI and NSC content than an equivalent weight of lucerne chaff, with wheaten chaff having the lowest GI of chopped hay roughages.**

Supplementation with the product Kohnke's Own TRIM™, which by way of its magnesium content has a role in normalising blood sugar by facilitating glucose use by the tissues and thus lowering blood sugar and its direct effect on the level of circulating insulin which peaks after a meal. TRIM also contains organic chromium which has a role in mimicking the action of insulin in response to blood sugar elevation after a meal. It also contains choline and manganese which have roles in normal fat metabolism and regulation of fat build up in the crest and tail butt areas.

TRIM is best given at double doses for 5-7 days or until the 'crest' or tail butt deposits become soft, then reduce to a standard dose for another 5-7 days. After this time, it is a good idea to monitor the 'hardness' of the crest and tail butt area at 2-3 day intervals. **If they again become packed HARD with sugars and fat, then reintroduce TRIM at double doses, as outlined above, until the crest and tail butt soften.** The risk of a horse foundering due to EMS if the crest and tail butt area remains soft is much reduced.

The addition of a supplement containing calcium, trace-minerals, including organic zinc and chromium, and a full range of vitamins, such as **Kohnke's Own Cell-Provide®**, as well as adequate salts, such as **Kohnke's Own Cell-Salts™**, will help maintain water intake and make up the shortfalls of water soluble nutrients and salts leached out by soaking hay when soaked hay is provided as a roughage base. These supplements, along with TRIM as required to maintain a 'soft' neck, have a role in maintaining metabolic function and vitality in IR affected horses. Research is continuing on other specific supplements that may help maintain metabolic function in horses prone to IR in retirement.

Monitor your horse's body weight every 5-7 days, but do not starve your horse, and particularly a pony or miniature, so that it will rapidly lose weight. Rapid weight reduction due to starvation can trigger fat mobilisation and the onset of a condition referred to as Hyperlipaemia in certain bloodlines of horses and ponies, which if not recognised early, can be fatal within 10-14 days.

Low GI Feeds	Medium GI Feeds	High GI Feeds
Soyabean hulls	Soaked lucerne hay (lowest roughage)	Oats
Lupin hulls	Soaked grass and cereal Hay	Corn
Sugar beet pulp (non molasses)	Wheat bran	Barley
Lupin seeds	Wheat pollard	Triticale
Soyabean meal	Rice bran	Rice
Sunflower seeds	Millrun (small amounts <10% of ration daily)	Millrun (large amounts >10% ration daily)
Copra meal (small amounts)	Commercial Feeds:	Molasses
Vegetable oils	Low GI	Apples
Carrots - fresh	Happy Hooves®	Pears
Commercial Feeds:	Pryde's EasiSport®	Grass, clover and cereal Hay
Very Low GI	KER Low GI Cubes®	Commercial Feeds:
Speedi-Beet®	Hygain Ice®	High GI
Hygain Zero®	Pony Pellets	Micronised Feeds
Pryde's EasiFibre®	Barastoc Calm Performer®	Extruded Feeds
Maxisoy Low GI SuperFibre Pellets®	Coprice Cool Conditioner®	Mitavite Formula 3®
	KER Equi-Jewel®	Hygain Micspeed®
	Pryde's Rebuild®	Most ' Performance ' feeds for racing and working horses, including grain mueslis or textured feeds with molasses.
	Most ' Cool ' Commercial Feeds - except those based on millrun.	

The photo at right shows hard tail butt fat deposits on a pony that displays symptoms of Equine Metabolic Syndrome



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