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The role of diet in the prevention and management of several equine diseases☆

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ABSTRACT

Modern feeding and housing practices of horses are typically directed at achieving a high level of athletic performance. There are some unfortunate consequences including an increased incidence of disease. Some of these diseases can be directly linked to dietary practices, while in others diet contributes as an important co-factor. Breeding practices to select for specific traits have also inadvertently resulted in the preferential selection of horses with genetic mutations within several breeds. In several of these genetic disorders specific dietary management is required for affected horses to achieve an acceptable level of performance. Diseases in which diet has a significant influence are discussed including equine metabolic syndrome, laminitis, diseases attributed to deficiency of vitamin E and/or selenium, exertional myopathies, nutritional secondary hyperparathyroidism, hyperkalaemic periodic paralysis, and the developmental orthopaedic disease complex.

1. Introduction

Diet plays a pivotal role in both the genesis and the management of several common diseases of horses. Animals involved in modern horse activities commonly receive a diet that differs markedly from that consumed by horses during recent evolution of the species, where the diet was typically fibre-rich and low in starch and was consumed slowly and continuously with little day-to-day variability in feed type or quality (Janis, 1976; Durham, 2009). In contrast modern diets commonly include large high starch meals that are interspersed with variable amounts of roughage. Changes in the type and quality of feed can vary widely and may predispose horses to a range of problems.

Modern feeding practices are commonly associated with problems of the gastrointestinal tract. For example, sudden changes in diet have been identified as the most important risk factor in the development of abdominal pain (colic) in horses (Archer and Proudman, 2006). The risk appears to be increased for up to 14 days after a diet change (Cohen et al., 1999). The sudden, and typically accidental, consumption of a large amount of starch, often grain, has long been identified as a cause of colic, abdominal bloat, diarrhoea, toxaemia, and laminitis. Diet has also been recognised as a contributing factor to several infectious intestinal diseases of horses, including salmonellosis and clostridiosis (Traub-Dargatz et al., 1990).

Abbreviations: AST, aspartate aminotransferase; Ca, calcium; CK, creatine phosphokinase; DE, digestible energy; DM, dry matter; DOD, developmental orthopaedic disease complex; EDM, equine degenerative myeloencephalopathy; EMND, equine motor neuron disease; EMS, equine metabolic syndrome; FFA, free fatty acid; GYS1, glycogen synthetase enzyme; HYPP, hyperkalaemic periodic paralysis; IR, insulin resistance; MJ, megajoule; NSC, non-structural carbohydrates; Na, sodium; NSH, nutritional secondary hyperparathyroidism; OC, osteochondrosis; P, phosphorus; PSSM, polysaccharide storage myopathy; PTH, parathyroid hormone; QH, Quarter Horse; RER, recurrent exertional myopathy; RYR1, ryanodine receptor; WMD, white muscle disease; WSC, water-soluble carbohydrate.

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Continuous access to pasture is considered to be protective with the incidence of colic less in this population than in horses that are predominately stabled (Hudson et al., 2001). Horses at pasture are not immune from colic and can develop problems with hindgut fermentation when large amounts of fructan-rich pasture are consumed (AlJassim and Andrews, 2009). Overstocking in sandy regions commonly leads to consumption of sand and an associated risk of intestinal obstruction and colic (Ragle et al., 1989).

The focus of this review is examination of the relationship between diet and several important diseases of horses. These include the equine metabolic syndrome (EMS), laminitis, diseases attributed to deficiency of vitamin E and/or selenium, exertional myopathies, nutritional secondary hyperparathyroidism, hyperkalaemic periodic paralysis (HYPP), and the developmental orthopaedic disease (DOD) complex.

2. Equine metabolic syndrome

A disease with similarities to human metabolic syndrome is recognised in the horse and is termed equine metabolic syndrome (Frank et al., 2010). Obesity and insulin resistance (IR) are factors shared by both syndromes (Johnson et al., 2006). The presence of, or history of laminitis is also characteristic of the EMS phenotype (Treiber et al., 2006). Hypertriglyceridaemia (Frank et al., 2006), hyperleptinaemia (Cartmill et al., 2003), arterial hypertension (Bailey et al., 2008), increased systemic markers associated with obesity (Vick et al., 2007), and altered reproductive cycling (Vick et al., 2006) are also associated with EMS.

The domestication of horses led to feeding practices that often differed significantly from the diets consumed in a natural state. Before domestication the genetic predisposition of "thrifty genes" conferred an evolutionary advantage. The accumulation of fat, development of transient IR and a proinflammatory state was beneficial for survival during times of limited feed availability. These changes abated when fat stores were depleted, typically at the end of winter (Johnson et al., 2006). The subsequent domestication and propensity to overfeed horses has led to year round persistence of adipose tissue with continual IR and its associated consequences. Adipose tissue is hormonally active and produces adipokines and adipocytokines (Rasouli and Kern, 2008). More than 100 different adipokines have been identified and it is the inappropriate secretion of these products over time that results in the pathophysiological consequences of obesity (Hutley and Prins, 2005). Specific adipokines that have been implicated in EMS include leptin, adiponectin, and resistin. Adipocytokines released by the adipose tissue or from macrophages within fat are pro-inflammatory and lead to a chronic state of low-level inflammation (Wisse, 2004; Vick et al., 2007; Rasouli and Kern, 2008). Similar to humans it is suspected that specific regions of adipose tissue in horse may be more hormonally active than other regions. One area is the accumulation of adipose tissue in the crest of the neck. A neck crest scoring system (the cresty neck score) has been developed to help distinguish horses that have developed regional rather than generalised obesity (Carter et al., 2009).

Insulin resistance is caused by defective insulin signalling at the cellular level leading to defects in a range of insulin-dependent metabolic and vascular processes, including insulin-mediated glucose transport (Kashyap and Defronzo, 2007). There is a compensatory increase in insulin secretion from the pancreas. As described earlier obesity and IR are clearly linked in EMS (Frank et al., 2006; Treiber et al., 2006; van Weyenberg et al., 2008). The link between IR and obesity may be due to the adipokine- or adipocytokine-induced down-regulation of insulin signalling pathways and/or the accumulation of intracellular lipids in insulin-sensitive tissues, such as skeletal muscle, a process termed lipotoxicity (Slawik and Vidal-Puig, 2006). Horses like humans vary in their genetic ability to develop IR; for this reason some obese horses do not exhibit IR (Frank, 2009).

The common signalment of the equid affected with EMS is the horse or pony between 5 and 15 years of age. The most common clinical signs at presentation include laminitis and prolonged generalised or regional obesity. It has recently been shown that supraphysiologic hyperinsulinaemia can induce laminitis in horses (Asplin et al., 2007). It is postulated that physiologic hyperinsulinaemia is a significant trigger factor in the development of laminitis (Johnson et al., 2010). The underlying effect of high insulin levels on the sensitive laminae tissues of the hoof is yet to be fully elucidated, but is suspected to be mediated by disruption to insulin-mediated vasoregulatory properties (Frank et al., 2010). It is reported that IR promotes a state of vasoconstriction due to decreased endogenous production of nitrous oxide (Muniyappa et al., 2007).

The diagnosis of EMS requires consideration of the patient signalment, history, physical examination including body scoring or neck crest measurement, and appropriate laboratory screening tests. Radiographic evaluation of the feet may also be indicated. The laboratory diagnosis of EMS is typically centred on the single measurement of blood insulin and glucose concentrations. Testing should be performed between 8 am and 10 am in the morning after a minimum of 6 h of grain and pasture deprivation. A small amount of highly structured carbohydrate can be fed and is useful in horses that become stressed with feed deprivation (Frank, 2009).

Horses with EMS tend to have a resting blood glucose concentration in the upper end of the normal range rather than being hyperglycaemic. Type 2 diabetes mellitus should be considered in older horses with persistently elevated resting glucose when external influences cannot account for the increased glucose. A serum insulin concentration greater than 20 μU/mL is consistent with IR, although there may be variability between laboratories (Frank et al., 2010). Test specificity can be affected in a variety of situations; false positives may occur when animals are stressed or experiencing pain, such as those experiencing laminitis. Testing should only occur when the animal is acclimatised to the environment and is without lameness or concurrent disease. Sensitivity of the test is affected when the horse is in the early stages of the disease or if pancreatic insufficiency has occurred due to cell exhaustion resulting in diabetes mellitus type 2 (Frank, 2009; Frank