



THE CARBOHYDRATE METABOLISM OF THE HORSE

FUNCTION AND DYSFUNCTION

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In horses like in other mammals, GLYCAEMIA (which is the concentration of glucose in plasma) is mainly regulated by two “antagonistic” and reciprocally-secreted pancreatic hormones: insulin and glucagon. These two hormones are the keystones of the regulation of the uptake, disposal and utilisation of fuel substrates by the body. In particular, they aim at maintaining glycaemia within a narrow, life-compatible range, throughout time.

For the horse, this normal range is approximately from 4.1 to 6.4 mmol/L (0.74 – 1.15 g/L).

Insulin is secreted by the beta-cells of the pancreatic Islets of Langerhans and has a hypoglycaemic effect. Its secretion is triggered by the ingestion and digestion of food, followed by the absorption of glucose and other metabolites. Its main effects are to inhibit glucose formation in the liver, and to allow



mones such as catecholamines and cortisol, growth hormone, leptin, progestagens, ...) (Geor, 2013).

The rate and extent a particular food induces changes in glycaemia can be expressed by the glycaemic index. It is a classification of feeds relative to their capacity to raise blood glucose, generally expressed as a percentage of the area under the curve response of glycaemia to a standard quantity of a test feed, compared to that of a standardised reference (whole oats in many studies concerning horses) (Hoffman, 2013). The same index can be built for insulin production, and research suggest that insulin response could be more relevant to consider than glycaemic response in the context of insulin resistance (Hoffman, 2013). After a meal, these patterns depend on several factors including the effects of meals consumed in the prior few hours (responses to grain-based meals in the afternoon are lower than in the morning, Gordon and McKeever, 2005), the size of the meal, the proportion of nutrients in the meal and in particular the starch and sugar content, the pre-caecal starch digestibility and the rate of ingestion (Harris & Geor, 2009).

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glucose to enter muscles and adipose tissue to be stored. In skeletal muscles, it promotes the synthesis of glycogen, a storage form of glucose, which is an important source of energy during exercise. From a general point of view, insulin has an anabolic action on the metabolism, of carbohydrates, but also of lipids and proteins.

On the contrary, glucagon has a hyperglycaemic effect. In the post-absorptive state, insulin secretion is reduced and glucagon secretion is enhanced, which results in stimulation of catabolic processes including mobilisation of glucose and fatty acids (Moore *et al.*, 2003). The same metabolic adaptations also occur during exercise. Glucagon is secreted by the alpha-cells of the pancreatic islets.

In addition to glycaemia, the release and effects of these hormones can be modulated by many other factors, including age, breed, pregnancy, lactation, nutrition score, diet, exercise, and other hormones ("stress" hor-

The glycaemic and insulin responses are much smaller when horses are fed roughage (e.g. preserved forage) when compared to grain-based feeds, although moderate increases in insulin production have been observed after ingestion of hay with relatively high content of non-structural carbohydrates (Borgia *et al.*, 2011). However, the addition of fibres to a grain-based meal does not substantially alter postprandial glycaemic and insulin responses (Vervuert *et al.*, 2009b ; Vervuert *et al.*, 2009c).

GRAIN-BASED MEALS, WITH HIGH GLYCAEMIC INDEXES, HAVE BEEN ASSOCIATED WITH:

- colic (Hudson *et al.* 2001)
- laminitis (Pass *et al.* 1998)
- gastric ulcers (Murray 1994)
- developmental orthopedic disease (Kronfeld *et al.* 1990, Ralston 1996),
- insulin resistance (Hoffman *et al.* 2003, Treiber *et al.* 2005) and
- polysaccharide storage myopathy (Valentine *et al.* 2001, Ribeiro *et al.* 2004).

The adverse effects of such meals are thought to be due to their high **starch** content (Hoffman, 2013). An excess in starch, sugars and/or fructans may promote or exacerbate these disorders, and still other metabolic disorders in the pathogenesis of which insulin resistance is thought to be involved: obesity, equine metabolic syndrome, EMS, and pituitary pars intermedia dysfunction, PPID, (also called Cushing's disease (Hoffman, 2013). Primary disorders of pancreatic endocrine function, like type I diabetes, are rare in horses. But abnormal sensitivity of peripheral tissues to insulin has been well documented in the last few years.

Insulin resistance represents a state in which the normal concentration of insulin fails to orchestrate a normal biological response, usually with reference to insulin-mediated glucose disposal (Kahn, 1978). In other words, normal concentrations of insulin fail to adequately increase the disappearance of glucose from plasma. However, excess insulin production usually occurs in response to insulin resistance, and allows glycaemia to be maintained within normal limits in most of the cases.

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Different conditions are thought to be associated with insulin resistance in horses, among which obesity, hyperlipaemia, EMS, laminitis, PPID, and osteochondrosis (Firshman and Valberg, 2007; Hoffman, 2013). On the contrary, other diseases like Polysaccharide Storage Myopathy (PSSM) or Equine Motor Neuron Disease (EMND) are thought to be associated with an increased sensitivity to insulin (Firshman and Valberg, 2007).

This association between insulin sensitivity and major pathological equine conditions has generated considerable interest in the study of factors influencing insulin sensitivity in horses. Unfortunately, these studies are impaired by the fact that the objective measurement of insulin sensitivity is very challenging (Firshman & Valberg, 2007). It has been shown that training increases whole-body insulin sensitivity (Stewart-Hunt *et al*, 2006). The extent of this improvement depends on the duration and intensity of training. The role of diet composition on insulin sensitivity is controversial. Some studies reported that feeding a high starch and sugar complementary feed (along with forage) decreased sensitivity in horses (Hoffman *et al*, 2003; Treiber *et al*, 2005) except if they were trained (Pratt *et al*,

2006). Treiber and collaborators (2006) suggested:

Insulin resistance develops with chronic adaptation to meals of grain and molasses, probably from the cumulative effects of repeated large fluctuations in glycaemia and insulin production after such meals.

Results of this study also suggested that insulin resistance may develop as a chronic adaptation to gradually increasing starch content in spring pasture. On the contrary, more recent studies found no effect or even an improvement of glucose disposal after adaptation to higher starch and sugar diets (Pagan



et al, 2011; Gordon *et al*, 2011). Conflicting results also exist for the effect of dietary oil on insulin sensitivity (Hoffman *et al*, 2003; Pagan *et al*, 2011). Age, breed and obesity also alter insulin sensitivity (Geor, 2013). Harris and collaborators (2006) suggested that daily physical activity, reduction of body condition score, avoidance of large meals of grain-molasses feeds, administration of antioxidants, and administration of (omega-6) and (omega-3) fatty acids favour insulin sensitivity, at least in healthy horses.

Due to the potential adverse effects of high responses in insulin, actual recommendations for healthy horses feeding are to limit starch

intake by 2 g/kg bodyweight (BW)/meal (Hoffman, 2013).

Other authors suggest even more restricted intake, (1,1 g/kg BW/meal), based on the moderate glycaemic (increase from ~5 to ~7 mmol/l) and insulinemic (increase from ~5 μ U to ~50 μ U/ml) responses observed below this threshold (Vervuert *et al*, 2009a). Borgia *et al* (2011) recommend that laminitis-prone horses and ponies should be fed hay with non-structural carbohydrates content <10–12 % to avoid post-feeding increases in circulating insulin concentrations that may increase risk of laminitis episodes. Similarly, it is sug-





BIBLIOGRAPHY

- Borgia, L., Valberg, S., McCue, M., et al. 2011. Glycaemic and insulinaemic responses to feeding hay with different non-structural carbohydrate content in control and polysaccharide myopathy-affected horses. *Journal of Animal Physiology and Animal Nutrition (Berlin)* 95, 798–807.
- Firshman, A.M., Valberg, S.J., 2007. Factors affecting clinical assessment of insulin sensitivity in horses. *Equine Veterinary Journal* 39, 567–575.
- Geor, R.J., 2013. Endocrine and metabolic physiology. In : Geor R.J., Harris PA and Coenen M, *Equine Applied and Clinical Nutrition*, 1st ed. Saunders, 33–63.
- Gordon, M.E., McKeever, K.H., 2005. Diurnal variation of ghrelin, leptin, and adiponectin in Standardbred mares. *Journal of Animal Science* 83, 2365–2371.
- Gordon, M.E., Jerina, M.L., Raub, R.H., et al. 2011. Insulin sensitivity in growing horses fed a higher starch versus a higher fat diet for two years. *Journal of Equine Veterinary Science* 31, 277–278.
- Harris PA, Bailey SR, Elliott J, Longland A. Countermeasures for pasture-associated laminitis in ponies and horses. *J Nutr.* 2006; 136:2114S–21S
- Harris, P.A., Geor, R.J., 2009. Primer on dietary carbohydrates and utility of the glycemic index in equine nutrition. *Veterinary Clinics of North America Equine Practice* 25, 39–50.
- Hoffman, R.M., 2013. Carbohydrates. In: Geor R.J., Harris PA and Coenen M, *Equine Applied and Clinical Nutrition*, 1st ed. Saunders, 156–167.
- Hoffman, R.M., Boston, R.C., Stefanovski, D., et al. 2003. Obesity and diet affect glucose dynamics and insulin sensitivity in Thoroughbred geldings. *Journal of Animal Science* 81, 2333–2342.
- Hudson, J.M., Cohen, N.D., Gibbs, P.G., et al. 2001. Feeding practices associated with colic in horses. *J Amer Vet Med Assoc* 219, 1419–1425.
- Kahn, C.R., 1978. Insulin resistance, insulin sensitivity, and insulin unresponsiveness: a necessary distinction. *Metabolism* 27, 1893–1902.
- Kronfeld, D.S., Meachem, T.N., Donoghue, S., 1990. Dietary aspects of developmental orthopedic disease in young horses. *Vet Clin N Amer Equine Pract* 6, 451–466.
- Moore, M.C., Cherrington, A.D., Wasserman, D.H., 2003. Regulation of hepatic and peripheral glucose disposal. *Best Practice & Research Clinical Endocrinology & Metabolism* 17, 343–364.
- Murray, M.J., 1994. Gastric ulcers in adult horses. *Compend Contin Ed Pract* 16, 792–797.
- Pagan, J.D., Waldrige, B.M., Lange, J., 2011. Moderate dietary carbohydrate improves glucose tolerance and high dietary fat impairs glucose tolerance in aged Thoroughbred geldings. *Proceedings of the American Association of Equine Practitioners* 57, 192 (abstract).
- Pass, M.A., Pollitt, S., Pollitt, C.C., 1998. Decreased glucose metabolism causes separation of hoof lamellae in vitro: a trigger for laminitis? *Equine Vet J* 26, 133–138.
- Pratt, S.E., Geor, R.J., McCutcheon, L.J., 2006. Effects of dietary energy source and physical conditioning on insulin sensitivity and glucose tolerance in Standardbred horses. *Equine Veterinary Journal* 36 (Suppl.), 579–584.
- Ralston, S.L., 1996. Hyperglycemia/hyperinsulinemia after feeding a meal of grain to young horses with osteochondritis dissecans (OCD) lesions. *Pferdeheilkunde* 12, 320–322.
- Ribeiro, W., Valbery, S.J., Pagan, J.D., et al. 2004. The effect of varying dietary starch and fat content on creatine kinase activity and substrate availability in equine polysaccharide storage myopathy. *Journal of Veterinary Internal Medicine* 18 (6), 887–94.
- Stewart-Hunt, L., Geor, R.J., McCutcheon, L.J., 2006. Effects of short-term training on insulin sensitivity and skeletal muscle glucose metabolism in standardbred horses. *Equine Veterinary Journal* 36 (Suppl.), 226–232.
- Treiber, K.H., Boston, R.C., Kronfeld, D.S., et al. 2005. Insulin resistance and compensation in Thoroughbred weanlings adapted to high-glycemic diets. *Journal of Animal Science* 83, 2357–2364.
- Treiber, K. H.; Kronfeld, D. S.; Hess, T. M., et al. 2006. Evaluation of genetic and metabolic predispositions and nutritional risk factors for pasture-associated laminitis in ponies. *Journal of the American Veterinary Medical Association* 228 (10), 1538–1545.
- Valentine, B.A., Van Saun, R.J., Thompson, K.N., et al. 2001. Role of dietary carbohydrate and fat in horses with equine polysaccharide storage myopathy. *J Amer Vet Med Assoc* 219, 1537–1544.
- Vervuert, I., Voigt, K., Hollands, T., et al. 2009a. Effect of feeding increasing quantities of starch on glycaemic and insulinaemic responses in healthy horses. *Veterinary Journal* 182, 67–72.
- Vervuert, I., Klein, S., Coenen, M., 2009b. Effect of mixing dietary fibre (purified lignocelluloses or purified pectin) and a corn meal on glucose and insulin responses in healthy horses. *Journal of Animal Physiology and Animal Nutrition (Berlin)* 93, 331–338.
- Vervuert, I., Voigt, K., Hollands, T., et al. 2009c. The effect of mixing and changing the order of feeding oats and chopped alfalfa to horses on: glycaemic and insulinaemic responses, and breath hydrogen