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

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" hormones 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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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:

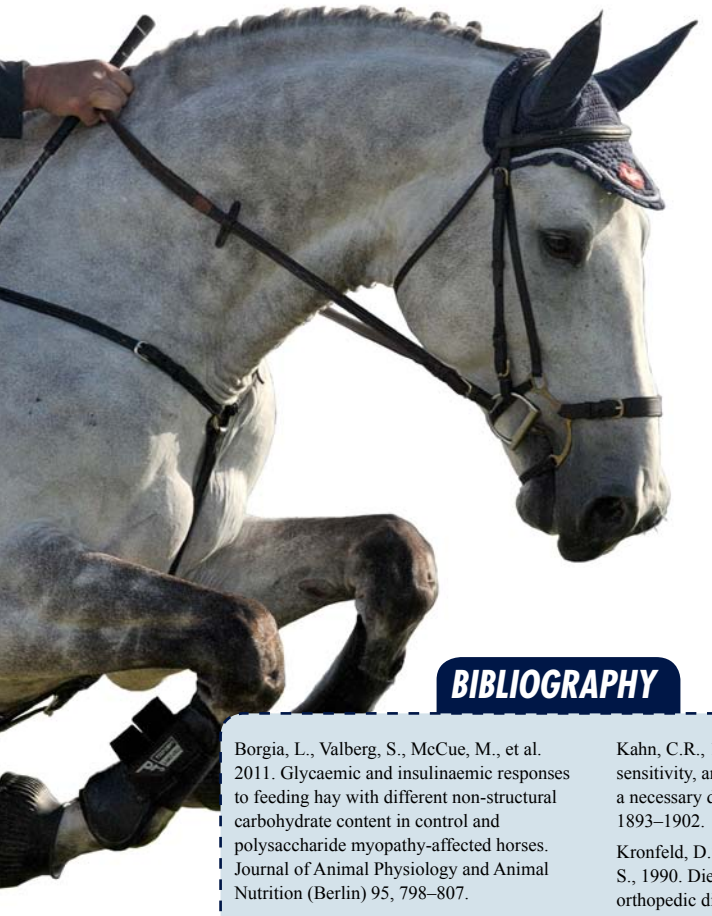
Insulin resistance develops with chronic adaptation to meals of grain and molasses, probably from the cumulative effects of repeated large fluctuations in glycaemia and insulin production after such meals.

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 µU to ~50 µU/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-



gested that starch intake should be limited to 0.3 g/kg BW/meal in horses and ponies suffering from metabolic disorder (Hoffman, 2013). However, more research is needed for definite feeding recommendations, in particular for sport horses, since their needs in non-structural carbohydrates are higher than sedentary horses, depending on their physical activity. Since the restoration of muscle glycogen content after exercise-induced depletion is particularly slow in horses the specific needs for performance, as well as for post-effort recovery, are to be further studied.

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