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Insulin Resistance:

WHAT IS IT, REALLY?

In horses as in humans, IR lies at the root of metabolic syndrome and type 2 diabetes

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Insulin Resistance (IR) is a commonly used and poorly understood term. Mostly, horse people associate it with hard cresty necks, fat deposits on the rump and shoulders, excess weight, Cushing's syndrome and the threat of a laminitic episode. Past these external signs and symptoms though, what is it really?

The concept of insulin resistance was first introduced in 1939 as a way to explain why cells become increasingly unable to use glucose in cases of diabetes mellitus (type 2 diabetes). Cells need to consume glucose to survive, and insulin provides the “push” for glucose to be taken up by cells. But researchers found that in type 2 diabetes, cells are less capable of taking up the glucose they need and that this inability is related to a decrease in insulin’s ability to move glucose into the cells.

So the term insulin resistance, now commonly called IR, was coined to describe the notion that in type 2 diabetes, there is a resistance to insulin-mediated glucose utilization. Insulin resistance has been studied intensively in human research over the last 70 years and is now understood to be the root of the metabolic syndrome that frequently results in type 2 diabetes.

GENETIC COMPONENT TO IR?

Equine research has demonstrated that the IR process is identical in horses—except they have the additional consequence of laminitis. Insulin resistance in both humans and horses starts the metabolic syndrome course, and it is considered to be the root of the disease process; in horses this process is generally referred to as Equine Metabolic Syndrome (EMS). It is widely accepted in human research that IR and EMS are genetically determined, and this is becoming increasingly more accepted in the equine research world. Understanding that IR is genetically activated explains why it so often cannot be controlled by diet and exercise alone.

It is important to understand that insulin resistance is an interactive process that escalates over time. Basically, this is how it works. Once insulin resistance starts, the horse’s body attempts to compensate for the cells becoming less “sensitive” to insulin’s action by increasing the amount of insulin produced. Insulin is secreted from the beta cells of the pancreas. When IR begins, these beta cells are called upon to secrete increasingly more insulin to make up for the resistance and get the needed glucose into the cells.

THE IR PROGRESSION CURVE

This compensation in the insulin secretion level by the beta cells occurs so the glucose level in the blood is maintained within the “normal” range. The assumption is that if the blood glucose level is “normal,” then the appropriate quantity of glucose is entering the cells.

The problem is that even though the glucose level is considered “normal,” the horse’s physiology, i.e., the beta cells of the pancreas, is now at a place where it takes a much higher concentration of insulin in the blood to push the same amount of glucose into a cell. At this point a horse will be diagnosed as having compensatory hyperinsulinemia (high blood insulin levels), and the risk of laminitis begins.

Some of these IR horses are going to reach a place where their pancreatic beta cells become exhausted and can no longer maintain the necessary “drive” to keep glucose within “normal” limits. For these horses, glucose becomes elevated in the blood and they may then be diagnosed as having type 2 diabetes, according to the classical definition of elevated blood glucose levels.

If blood glucose levels are high, i.e., above normal concentration, then the glucose available for cellular use is deficient and inadequate to meet the needs for their health. Every increase in



Hard, cresty necks are hallmarks of insulin resistance (IR) and equine metabolic syndrome (EMS), predisposing the horse to laminitis. Research has shown that the metabolic process is identical to that leading to type 2 diabetes in humans. Photo by Jill Willis/Courtesy AANHCP

the severity of insulin resistance and compensatory changes in organ function brings a greater risk for laminitis and associated pathologies.

The familiar signs of a horse at risk for laminitis—hard, cresty neck and fat deposits on the rump and shoulders—stems from this internal process. Once IR has begun, the consequences of the metabolic syndrome that are set in place include not only the increased risk of laminitis but also decreased immune function, vascular issues, a variety of categories of anemia and gastrointestinal problems.

Obese horses have IR and, contrary to popular opinion, non-obese horses also have IR; the external signs and symptoms are merely an “outside” expression of the progressive underlying disease process. “Proper” diet and exercise are necessary for these horses, just like it is for people with metabolic syndrome and diabetes, but diet and exercise will not reverse the course of this disease. Good/healthy care and management practices are an important part of decreasing the “trigger” aspect of an acute laminitic episode but are not always enough to prevent these damaging and painful experiences from occurring.

Laminitis is a consequence of the above-described disease process and not the disease itself. You may well be able to recover the damaged hoof, and you should, but if you don’t effectively treat the root issue of insulin resistance your horse will remain at high risk for another laminitic episode.

EXERCISE, HOOVES & NUTRITION

- ▶ Since virtually all IR horses have some degree of immune deficiency, exercise can be a double-edged sword. There should never be stall rest, and the horse should be “living” in an area where hay is placed around it (aka “Paddock Paradise”) so the horse needs to move to eat and drink water. One must monitor each horse’s progression along the IR curve with diligent observation and specialized blood work (For more informa-

continued next page



When your horse is healthy, his hooves are healthy! Educate yourself on the characteristics of the healthy, naturally shaped hoof—low heels, relatively short toes, a straight hoof wall free of prominent stress rings and a mustang roll or bevel around the hoof wall that allows your horse to move freely and comfortably. Photo by Bobbie Jo Weber

“Your horse does not need an external trigger such as carbohydrates or fructans to have a laminitic episode. Heat with or without humidity amplifies the risk of damaging consequences.”

tion, see my article, “A Simple Lesson in Blood Chemistry for the EMS/IR Horse” here: http://www.forloveofthehorse.com/blood_chemistry_analysis.php

- Keep your horse on a schedule of regular, balanced trimming to keep heels low and toes back to maintain tight laminar connections and proper function. Be sure to maintain a good mustang roll on the hoof, as long toes will put more strain on the laminae as the foot breaks over.
- Test each new load of grass hay (avoid alfalfa) to determine levels of sugar and starch—optimally these levels should be lower than 10%. Do not feed your horse carrots, apples or anything sugary.



Joseph Thomas, PhD, has been a practitioner, teacher and consultant in Chinese medicine for more than 25 years. In addition to his work as a Massachusetts Institute of Technology (MIT) scientist in medical research, Dr. Thomas apprenticed with Leon Hammer, MD, one of the world’s foremost experts in diagnostics and Chinese herbalism. He has united these skills with his love of horses to create an extensive selection of proprietary Chinese herbal formulations. He and his wife, Crystal Leaman, developed For Love of the Horse, a natural horse care company dedicated to providing clinical services and Dr. Thomas’ precisely blended herbal solutions. Visit www.forloveofthehorse.com



Seasonal Triggers for Insulin Resistance

High Heat and/or Humidity

Summer heat and humidity dramatically exacerbates the signs and symptoms of IR, which of course includes diabetes, since this is the end result of IR. What does this mean for your IR horse? Your horse DOES NOT need an external trigger such as carbohydrates or fructans to have a laminitic episode. Heat with or without humidity amplifies the risk of damaging consequences.

Why does this happen? High heat increases metabolic rate; this includes digestion. An important consequence of this aspect to an IR horse is there is significantly more hydrochloric acid produced and secreted into the stomach. This will lower pH, thereby increasing intestinal acidity, which enhances the risk of colic and/or a laminitic incident. The latter consequence is amplified above the already-low pH of an IR horse because of their proclivity towards biliary interference, which lowers intestinal pH. Lamina stretching is very common for horses in summer heat, even with those who have not had any problems in the months leading up to this heat.

Endocrine metabolism is increased as well with the effects of greater liver and pancreatic endocrine activity. This means the liver will produce more glucose and secrete this excess into blood circulation and transform excess glucose into glycogen for transport for storage to fat and muscle tissue, making fat deposits—such as a cresty neck—more prominent. A horse will be more likely to tie up or just be stiffer, especially in the hind end.

Pancreatic beta cells will secrete more insulin into the blood, thereby raising insulin values above the already hyperinsulinemic level your horse was at going into the heat.

All of this increases laminitis risk.

Effects of Cold Temperature

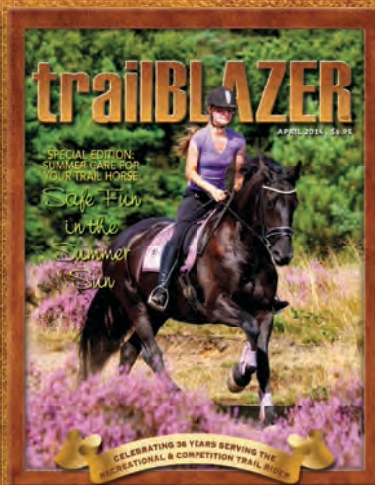
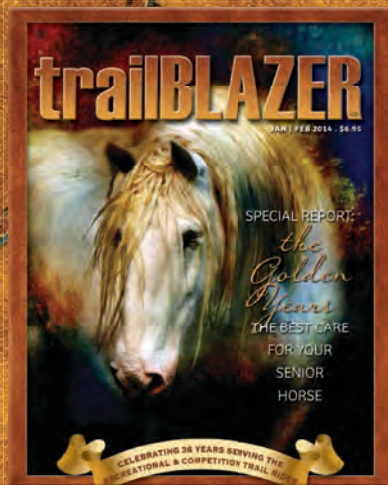
At the other extreme, cold temperatures set off seasonal endocrinologic variations, but in the cold there is a direct effect upon the adrenal glands. The adrenal cortex secretes cortisol and responds to the cold directly through an increase in insulin resistance; i.e., hyperinsulinemia (elevated serum insulin), which is directly related to the liver’s increase in glucose production. The hormone 11 beta-HSD-type1 gets overexpressed through all of this intricate interacting, which must necessarily result in more glucose production. IR is the root culprit of the winter’s exacerbation of our horse’s increased risks in this season because it is hyperinsulinemia that signals the adrenal gland’s increased secretion of cortisol. Chinese medical theory enters here beautifully, because in this medical system, kidney yang “rules” the adrenal glands and easily becomes deficient in the winter since kidney is associated with this season, yang is “hot” and cold easily dampens its functions.

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