Feeding horses –horses need nutrients –feeds supply nutrients

The NRC determines nutrient requirements

No safety factor.

Energy is not a nutrient , it is the fuel for all metabolic activity.

Energy is derived from nutrients like fat and carbohydrates –Energy is the number one metabolic need for horses next to water. Other nutrient needs are sacrificed to provide energy if deficient.

Use ideal weight to determine energy needs.

Energy needs vary -Breed differences, Work level , environment , health issues. The type of exercise, temperament, reproduction and lactation or growth. Stress ? Must he gain or lose weight?

How do we feed properly?

Horses normally can derive all the energy and most of the other nutrients from native range which has a variety of plants and rely mostly on grasses .

A small amount of sugar and starch is normally found in forage and digested in the small intestine as in monogastrics like pigs or humans. Grass contains fructans as storage form of carbohydrate

Bacteria in the hind gut ferment fiber (the structural component in forage) to volatile fatty acids used directly by the horse for energy or stored as fat or changed to glycogen and stored for future use in adopose tissue and in muscle and the liver respectively.

The fiber is made up of glucose units joined together to form cellulose and hemicellulose and pectin.The fiber is stiffened by a compound called liginin which protects some of the fiber from fermentation .

Forage is number one! Horses are hind gut fermenters designed to use fiber.

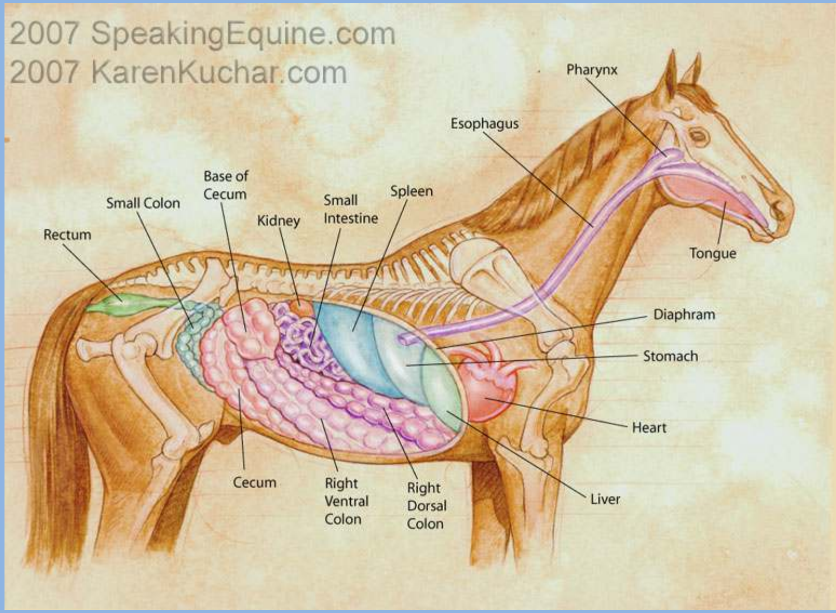
Forage is the natural food for horses . They are designed to eat grass!

Maximize hay/forage and pasture to mimic nature.

One must provide adequate forage to maintain the population of bacteria that ferment fiber to ensure a healthy gut and in turn a healthy happy horse.

Feed a minimum of 1.5% BW as forage.

Horses usually eat from 1.5-2.0% of BW dry matter as average quality forage. This can support energy needs for maintenance to light work.



Horses can eat more of forages lower in fiber .

Horses rely on eating high volumes of forages like native grasses . They eat little and often. They will graze 16-17 hours on native range, even getting up in the middle of the night to graze.

We need to Feed little and often to mimic nature.
appetite well.

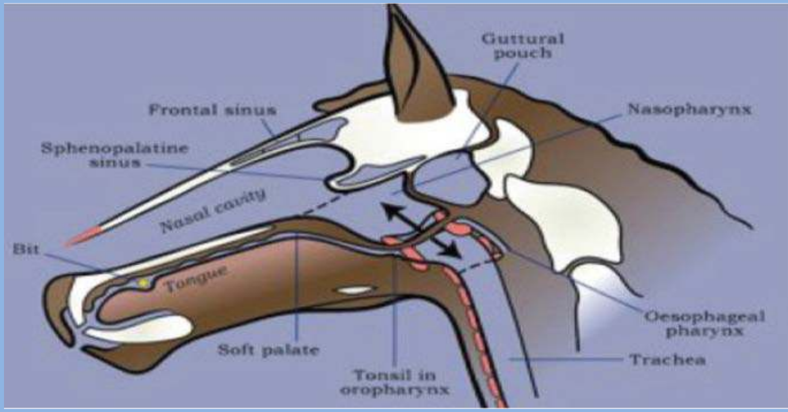
Chewing is critical

- Digestion starts with apprehension of grass , biting it off , secreting saliva , then Chewing and swallowing the moist bolus.

- -the jaw moves 60,000 sweeps/day on pasture .

  Forage promotes chewing which has many health benefits starting with breaking open the forage for digestion.

- Chewing stimulates saliva production which contains the buffer sodium bicarbonate which promotes health of the digestive tract . Chewing relieves boredom and prevents stereotypy behavior.



Chewing is critical

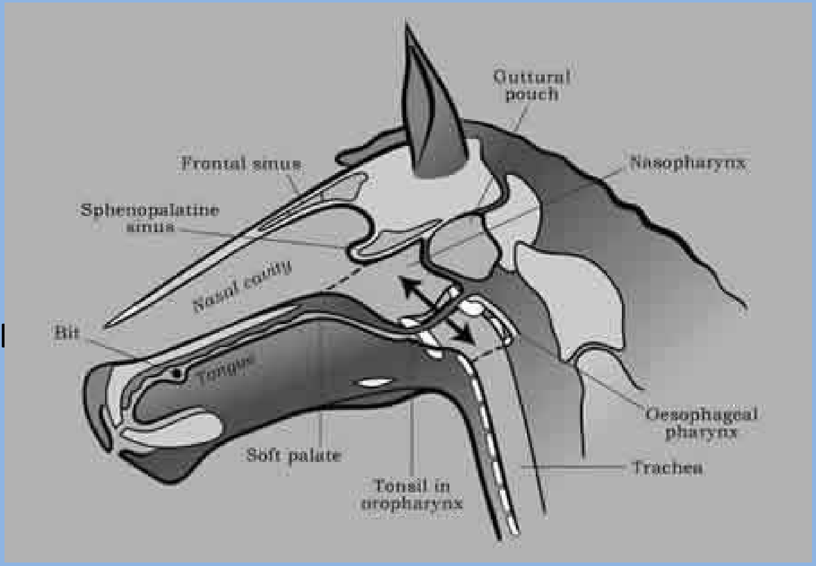
- Average Hay-
  Chew half hour per 2 lbs vs grain 10 minutes per 2 lb. Hay encourages much more saliva production than grain .

- Average Hay-4000 chews /kg vs grain 850 chews /kg

- Chewing grains is not a full jaw sweep so sharper edges on teeth. Seniors are less efficient at chewing.

- Choke may occur easily as horses have a cuff of tissues around the esophageal opening and must push feed up and over it with their tongue to swallow properly.

- Horses without feed do not produce saliva, but they do produce acid in the stomach 24/7. This can be a problem.



- Ulcers -5 hours rule vs natural grazing. Horses secrete acid 24/7 and feed does not stay in the small stomach very long. Horses allowed to get empty are vulnerable to acid damage in the stomach.

- A good level of fiber in forage to encourage chewing ,

- Extremely coarse forage may cause ulcers (new data).
- Horses will slow intakes of coarser forage and drop intakes of very coarse forage.

- Older horses may have loose teeth and once over 21 teeth stop erupting from below. From then on the teeth gradually lose their grinding ridges and digestion efficiency. (smooth mouth or marble mouth!).

- Ulcers -5 hours rule vs natural grazing. Horses secrete acid

- Minis “may” have issues chewing due to jaw allignment . They are also prone to impaction colic vs horses because their digestive tract is narrower.

- Adult horses may secrete up to 35-40 liters / day saliva with a pH of 8.6-9.1. This is very alkaline.

- Horses do not chew poor forage better!

- They do not chew rich soft low fiber forage well and may get acidosis , ulcers or gut issues. Failure to provide adequate chew factor will result in abnormal behavior such as wood chewing , eating dirt or manure or worse. They will self medicate if they lack fiber.

Hind gut health:

Over use of grains results in sugar and starch moving through the digestive system into the hind gut .

Bacteria that love sugar and starch then proliferate causing digestive upset and other metabolic problems

These sugar/fructan and starch loving bacteria produce lactic acid .

The pH may drop rapidly, killing off the friendly bacteria that ferment fiber, releasing endotoxin and biogenic amines and other harmful substances, and gut damage may occur allowing harmful compounds to be absorbed. “The leaky gut”.

Result inflammation , sepsis, possibly diarrhea and laminitis

Horse saliva is very low in amylase.

Maximum SS is 1-1.5 g/kg bw per meal

Two grain meals per day.

Max meal size now considered to be 1.5 kg vs 2.27 kg /meal

Max grain at 1.0% body weight. Overall feed intake 20-2.5% bwt.

.

Hind gut health:

The Saliva of horses only contains small amounts of the enzyme amylase for starch digestion, unlike humans. Partly why horses are not as efficient at digesting starch as other animals.

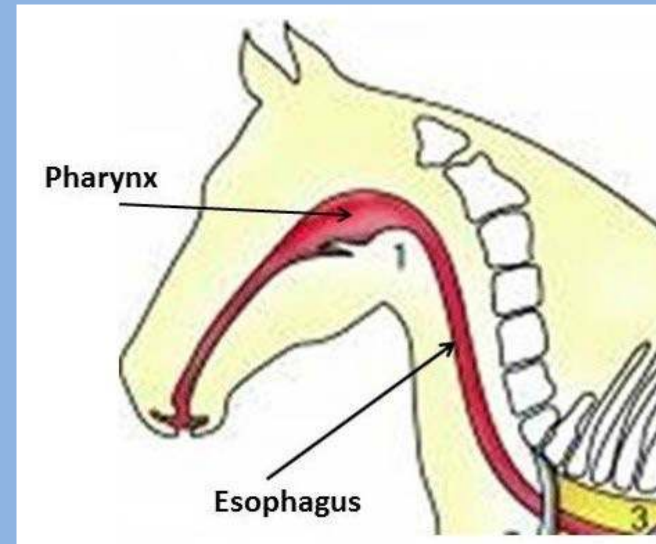
Hay encourages water intake at 3-5 to one Vs grains which may only have a 2-2.5 to one water intake vs dry matter consumption

Water and the forage ensure Bulk and ballast to the GIT holding it in place.

The hind gut also acts as a water and electrolyte reservoir.

Esophagus to stomach usually a one way system-

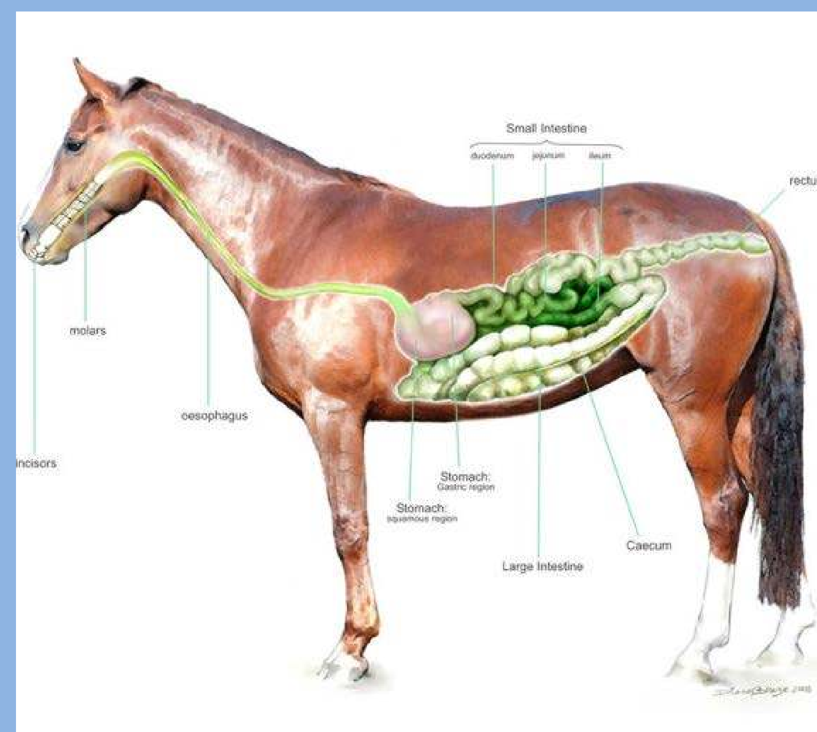
Esophagus 50-60 inches long . No mucous protection, can choke. Once food is swallowed it is a one way system. Horses cannot belch or vomit.



Horses are unique. Relatively small stomach at 8-12 liters or 8-9% of the GIT

They have evolved to eat little and often.

The stomach does not hold feed very long . Food may only stay 12-15 minutes.



Naturally never full, best if only 2/3 rds to maximum ¾ full. A layered effect with a mat of forage on top on pasture. Top alkaline , bottom very acidic.

Produces acid 24/7 to start protein digestion along with the enzyme pepsin, stops fermentation from the initial intake prevents gas colic.

Produces a small amount of gastric lipase to start fat digestion

Two areas –top (saccus caecus) has no protective mucous and bicarbonate secreting tissue lining. The pH is high (alkaline) in the top portion.

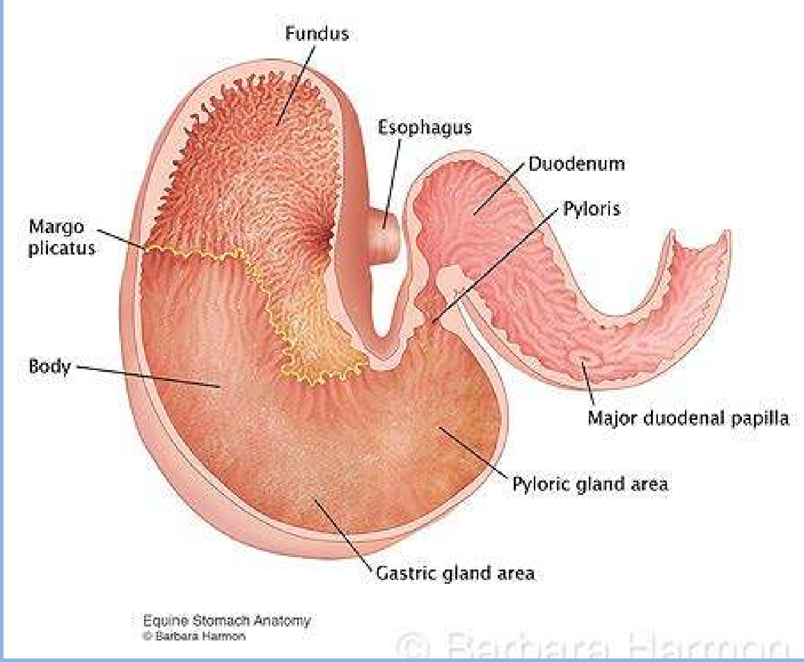
Lower portion (fundic and pyloric area) secretes acid and pepsinogen. Lined with mucous and cells secreting bicarbonate . Protected from strong acid. Very low pH towards the bottom.

Excess fermentation in the stomach may cause rupture as the esophageal valve and the pyloric sphincter do not allow gas to escape easily.

The stomach is also up inside the rib cage and it is hard to see swelling occuring.

Please call your vet ASAP at the signs of any discomfort and pain . Colic is the number one killer of horses.

Esophagus to stomach usually a one way system-Produces a small amount of gastric lipase to start fat digestion



This can be dangerous as the esophageal valve and pyloric sphincter do not allow gas to escape by belching or vomiting

Never turn an empty hungry horse out on rich spring grass.

(grass colic.). Provide some hay first before turn out.

Pasture is ideal for prevention of colic and ulcers if managed.

Feed hay before grain. Feed some alfalfa a natural buffer. Time the alfalfa .

Gastric Ulcers- follow vets advice on treatment.

70-90% of race horses

75% of 3DE horses

50-75% of western performance horses and

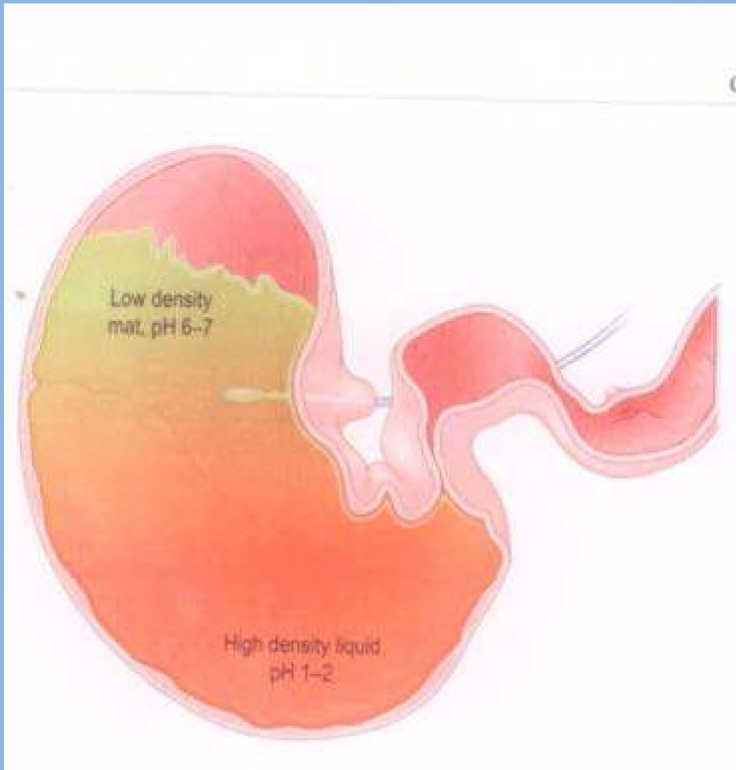
52% of dressage horses

10-30% of horses in 5 swiss barns ridden for pleasure .

10-30% of wild horses.

42-70% of foals.

The stomach-Fermentation may occur here when over feeding grains or due to consumption of rich grass pasture.



Stomach and performance

. E.G. Breathing matches stride rate up to approximately 120 breaths per minute and affects stomach health.

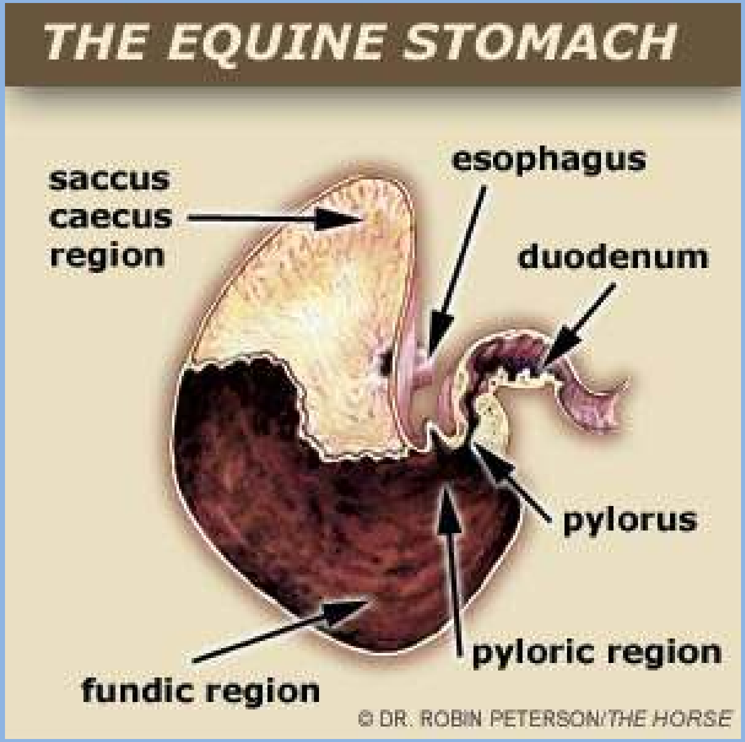
The upper section is vulnerable to acid pushed up during collection, and normal body compression during exercise, or cribbing.

Ulcers usually form along the Margo Plicatus /cardiac area which separates the protected from the unprotected zone.

Excitement of exercise , Stress hormones and pain hormones increase acid production.

Alfalfa is a natural buffer.

Ideal for enhancing energy for growth or performance . Consumed at a higher amount than grass hays of similar fiber content.



Stomach and performance

The “squirts”” with relatively normal fecal balls but water coming out after defecation is due to body compression due to mental stress and usually is found in the low horse in the pecking order.

Very coarse forage may damage the lining of the hind gut and causes ulcers Recent Danish research.

Back flushing of duodenal contents like strong bile salts and acids may happen if a horse is empty for a long time , creating ulcers.

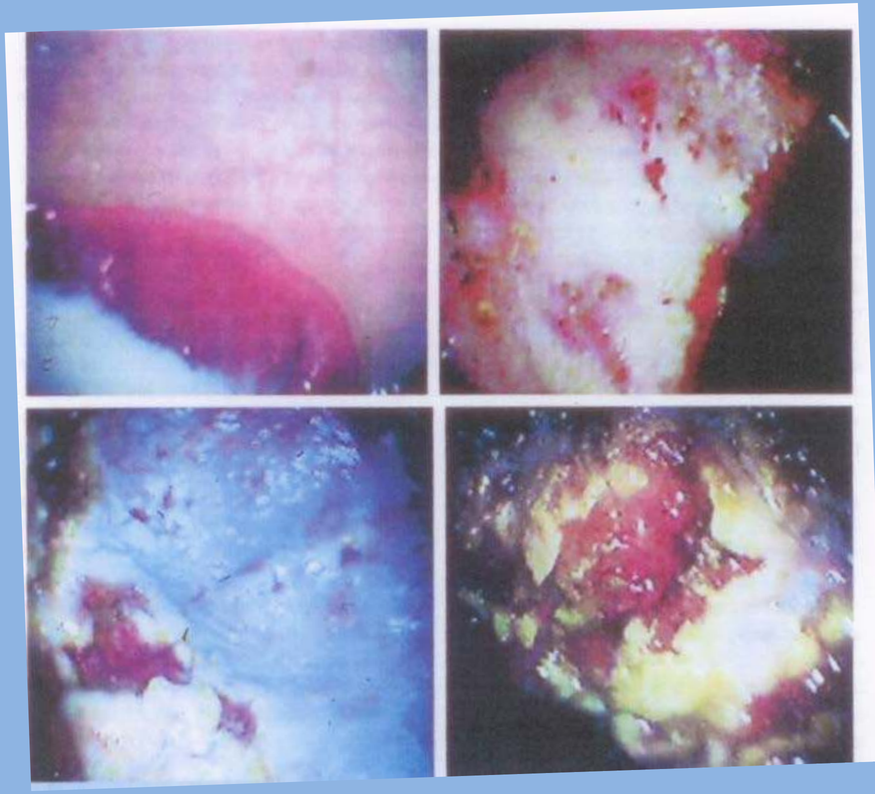
Lower lining-Protective mucous layer containing bicarbonate , PGE2 promotes the production mucus . Stimulated by essential fatty acids.

Hydration is critical. It dilutes acid, encourages proper digestion and movement in the GIT.

Metabolic adaptations to Exercise and ulcers

Shifts in fluid volume shunt blood away from the GIT and kidneys to the muscles for work and for temperature control to the skin.

May compromise gut health re colic in poorly hydrated horses. 15-20% decrease in plasma volume after only 4-1-minute steps of incremental work has been recorded.



Decreases in plasma volume after this point are due to losses of total body water as in sweating and evaporative loss due to heating effects. Result is a decrease in plasma volume further compromising circulation to the gut and kidneys . Heart rate goes up to compensate for lowered plasma volume due to poor hydration.

Horse sweat effects are different from humans . Their sweat is hyper tonic human sweat is hypo tonic. Horses do not get the metabolic signal to drink when dehydrated due to sweating.

Metabolic adaptations to Exercise and ulcers

The thirst response is also shut down by adrenaline. This is an evolutionary adaptation for escaping a predator.

EG suppression of drinking after endurance events.

Allow 2-4 l water per kg dry matter, increase 15-20% if warm weather. With prolonged exercise allow 300% or more to replace sweat losses.

Horses can lose 10-12 liters sweat per hour in extreme heat.

Suppression of water intake after exercise may occur with cold hypotonic water [passing over the nerves of the mouth) . It should be moderate in temperature and use electrolytes as well as plain water. Horses may drink less in cold weather and get impaction colic.

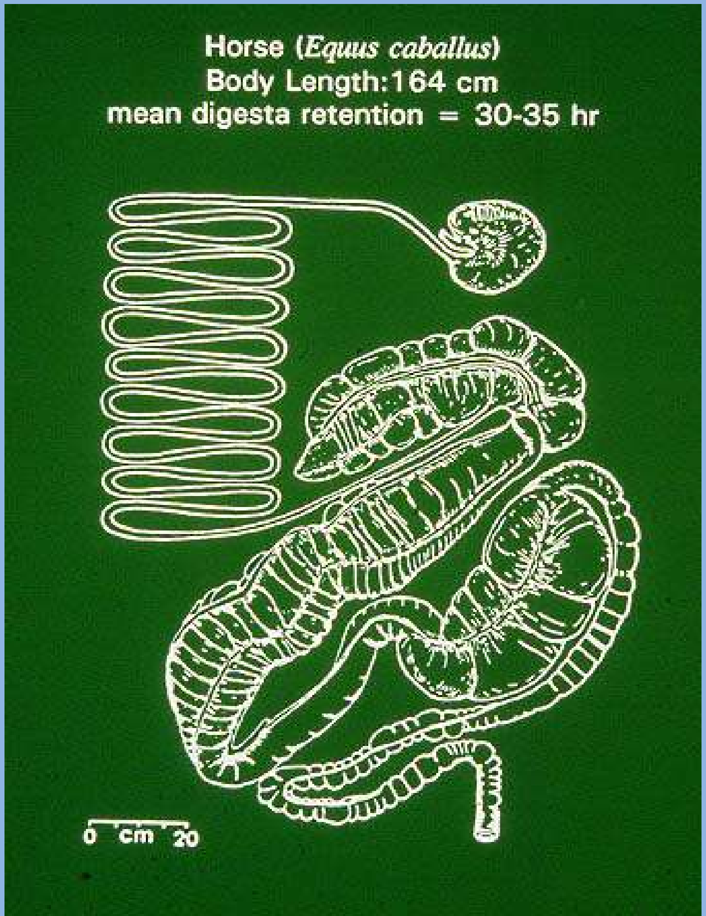
Endurance horses can be taught to drink warm water with electrolytes during competition.

Small intestine-25-28% of capacity, about 60-70 ft long. . Site of enzymatic digestion of CHO, Protein, and Fat, minerals/trace minerals and Vitamins.

. Site of enzymatic digestion of CHO, Protein, and Fat, minerals/trace minerals and Vitamins. Food rate of passage is only 30-90 minutes and partly why horses are less efficient at digesting starch. Starches are well broken down initially to glucose, but the glucose is not absorbed well. Glucose absorption is less efficient than humans.

Short time may limit starch digestion and may possibly be a limitation for glycogen reloading. Horses do not reload glycogen until 72 hours unlike other animals and humans who only need 24 hours. (hydration critical for glycogen reloading)

A portion of the small intestine is not anchored and vulnerable to twisting and displacement.



This enhances insulin production by 2-3 times that of a similar glucose load given IV.

This system seems to be more active in breeds prone to IR/ID (insulin resistance or insulin dysfunction) or just plain hyperinsulinaemia.

Incretins are the hormones secreted that stimulate the pancreas and may be a target for control of insulin. Already used for type two diabetics to enhance insulin production.

Small intestine New-The enetero-insular axis . Sensors in the small intestine detect glucose and other sugars and directly stimulate the pancreas to secrete insulin before glucose is absorbed and reaches the pancreas.

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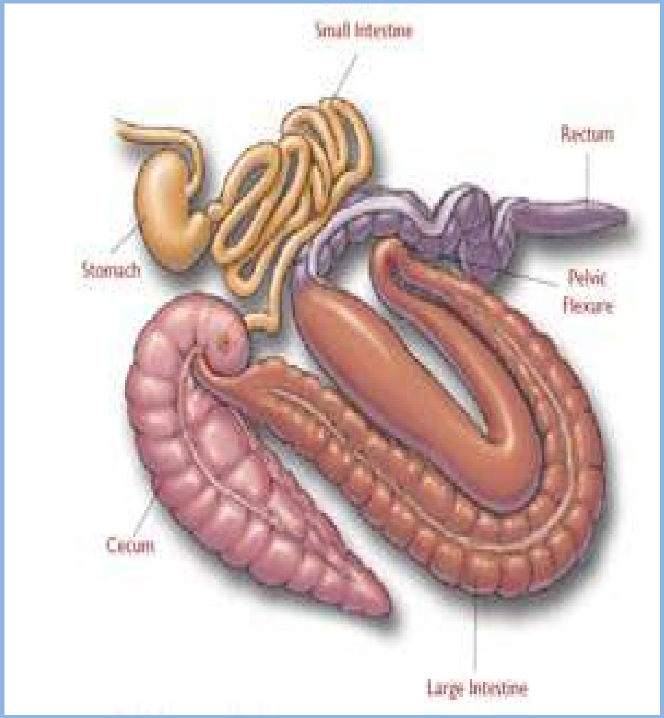


Enlargement to promote Microbial fermentation of plant fiber for Energy . Restrictions slow rate of passage to aid bacterial fermentation.

However, restrictions and folding are catch points for impaction colic

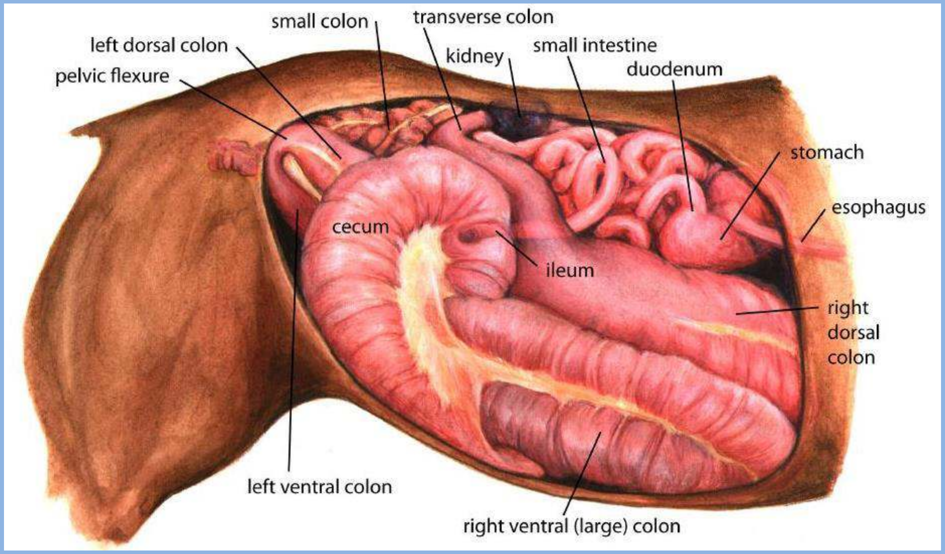
Large intestine:65% of capacity Cecum and colon. Cecum is a blind sac 30 l -feed stays about 7 hours. Easily impacted with poor feed or poor hydration, or poorly chewed feed as per seniors.

Limit starch and sugar to prevent spillover into the hind gut. Maintain the biome!

Ensure grain fed at least 6 hours apart

Max grain per meal 3.3- 5 lbs ? and max per day 10 lbs for a 1000 lb horse. Feed hay first.

Max is 1% of body weight as grain per day.



Microbial fermentation also produces

VFAs , B vitamins, Vitamin K, and C

Absorption in large intestine

A water and electrolyte reservoir (eg sodium potassium and chloride)

Phosphorus (Bacterial Phytases help with phytates)

Many twists and turns. A problem area. Water and electrolytes are absorbed here. Ideal pH 6-7

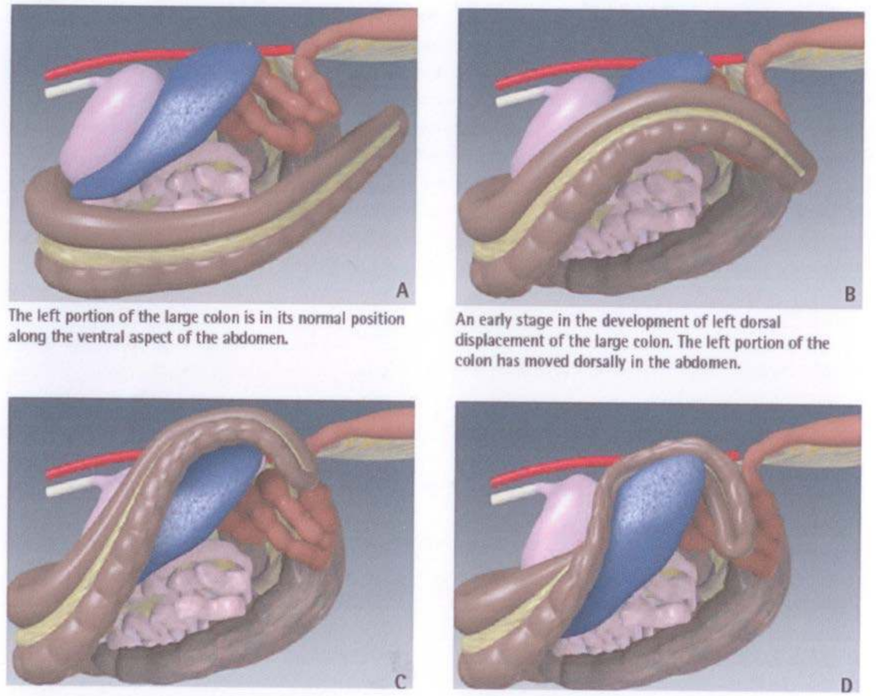
Digesta moves to small colon and fecal balls are formed here water is absorbed and the fecal balls are stored in the rectum

Fiber and water add ballast to prevent colic from displacement, twists and impaction.. Use some soluble fiber!

Large intestine-large colon-75 liters 12 feet long Microbial fermentation also produces

VFAs , B vitamins, Vitamin K, and C

Absorption in large intestine

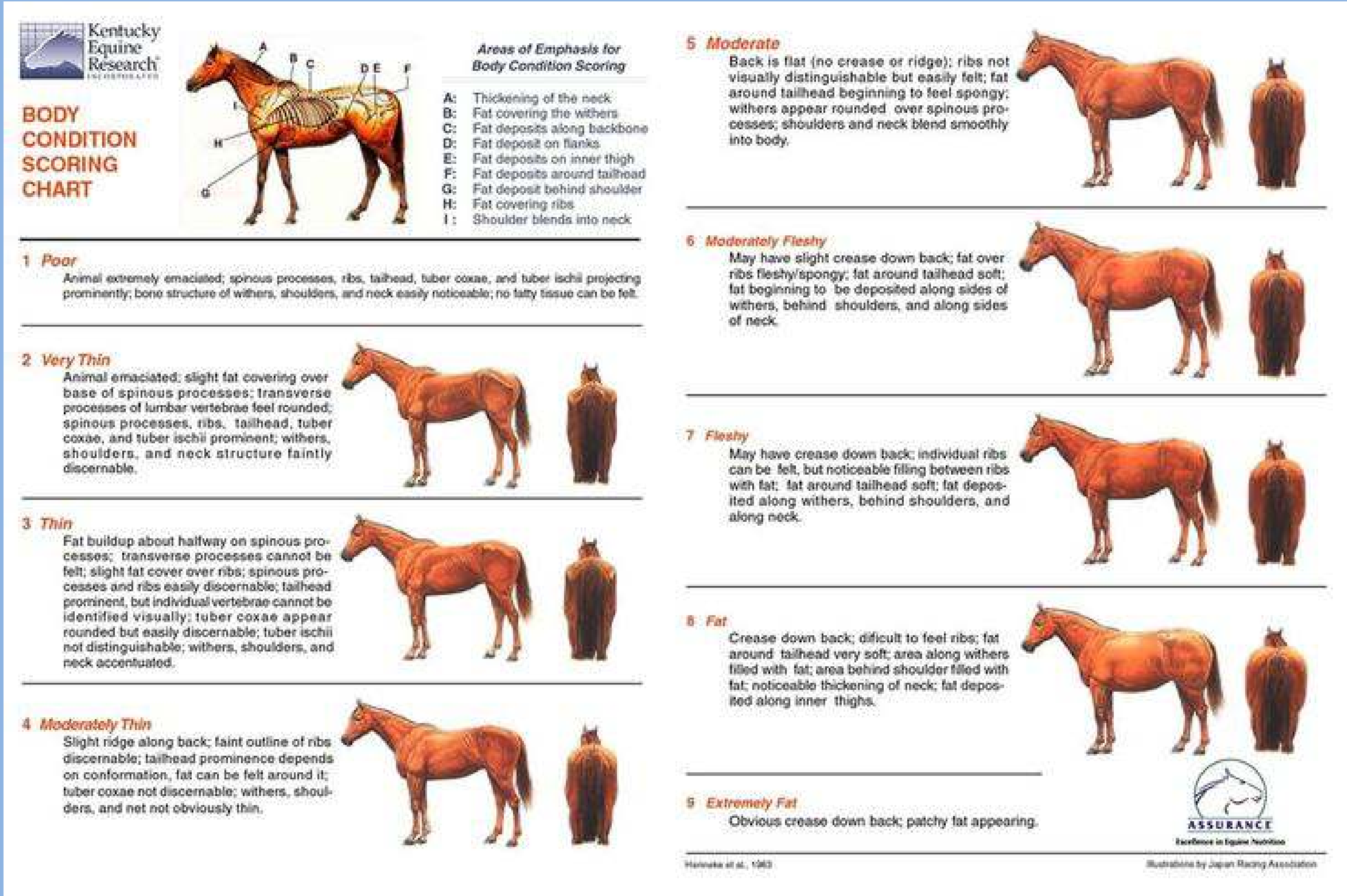


The left portion of the large colon is in its normal position along the ventral aspect of the abdomen.

An early stage in the development of left dorsal displacement of the large colon. The left portion of the colon has moved dorsally in the abdomen.

.vetfolio.com/internal-medicine/making-sense-of-large-colon-displacements-i.

Feed for body condition



Feed for good Body Condition-The Henneke Scoring system

A body condition of 5 is ideal. A condition over 5 is over weight. A condition over 7 is obese possibly leading to harmful endocrine and metabolic changes including insulin dysfunction /insulin resistance or high insulin and laminitis.

Losing condition below 4.5 is probably muscle mass loss.

Horses needing weight require approximately 9 Mcals per day per lb of gain .

Horses must be put on a diet if over weight but this is complicated as per digestive function, and the mental stress it puts horses under.

Exercise is important in the process.

Not all IR (insulin resistant)horses are obese

Not all obese horses are IR

Lean horses may be very IR or dysfunctional

Obese horses may be perfectly healthy

Genetics plays an important role as some horses may be high insulin just after a meal yet insulin sensitive?

EMS –the relationship between obesity and insulin resistance- laminitis -3 Criteria

1. Increased adiposity, generalized, regional, or both, with emphasis on Nuchal ligament/tail head /omental Adipsoe Tissue accumulation.
2. Peripheral insulin resistance/dysregulation.
3. A predisposition to laminitis in the absence of risk factors for other types of disease, such as sepsis or enterocolitis.

High insulin alone may trigger laminitis . New!

Other factors may be be included later. eg hyper leptinemia, seasonally increased Blood Pressure or altered reproductive cyclicity.

Horses with EMS are typically over weight to obese with BCS from 7.0-9.0/9.0

Horses with EMS may have pre-existing radiographic changes or abnormal rings on their hooves before the first recognized episode of laminitis. Keep your vet involved with hoof issues.

Feed for good Body Condition-The Henneke Scoring system

Laminitis due to EMS is often observed in at risk animals (i.e. Obese and/ or with Insulin Dysfuntion) exposed to a high NSC diet either at pasture (PAL) or as a supplemented diet.

The mechanism remains obscure.

EMS associated laminitis is unique associated with insidious onset and a variable degree of lameness.

Early signs are often missed , perhaps only abnormal hoof growth is seen.

Lamellar stretching but minimal lamellar separation is evident early in endocrinopathic (hormonal related) laminitis as per EMS or Cushing’s disease or excess cortisone.

There is relatively little inflammation.

Unique lamellar signalling may cause the lamellar disruption present in EMSAL. Laminitis !

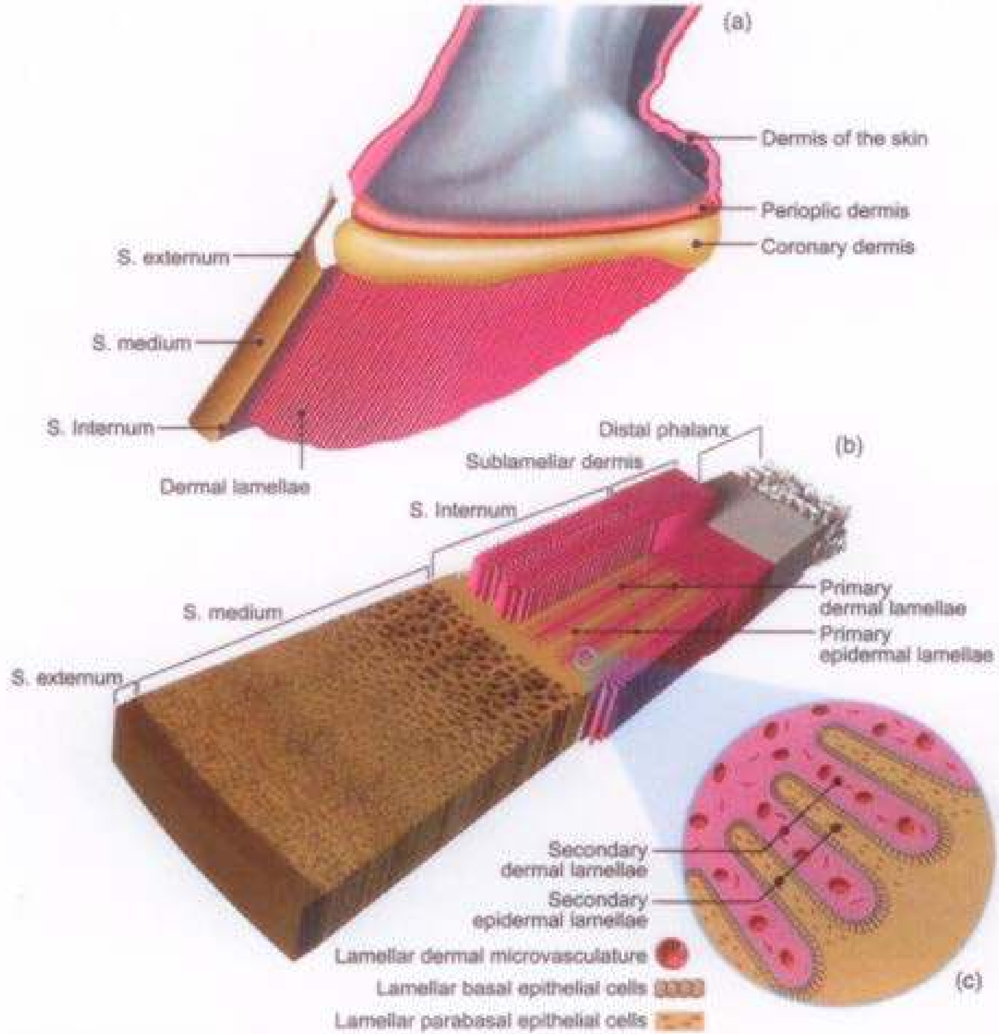


Figure 3.5 Schematic diagram of the anatomic relationship between the hoof capsule, the digital lamellae, and the distal phalanx. The parietal integument responsible for suspension of the distal phalanx within the hoof wall. Note the primary dermal lamellae (A) which interdigitate with the epidermal lamellae of the stratum internum (B), there being approximately 550–600 of each. Note also, on a 'slice' of the digit in a frontal plane (B and C), the increased surface area of the interface between the dermal and epidermal lamellae due to interdigitation of the secondary epidermal and dermal lamellae. Suspension of the distal phalanx within the hoof wall is dependent on the attachment of the lamellar basal epithelial cells (C) to the underlying basement membrane continuous with the lamellar dermis. Illustration courtesy of Tim Vojt, The Ohio State University; The Ohio State University.

Insulin

- Insulin resistance-Insulin dysregulation (ID) is a better term

- ID-covers insulin resistance and both persistent or intermittant hyper insulinemia.

- Horses can have excessive insulin response to peripheral insulin resistance.

- Recent research on the entero-insular axis shows horses can have an excessive insulin response to ingested carbohydrates without concurrent insulin resistance which results in a transient hyperinsulinemia . This axis is made up of two cell types that sense the presence of carbohydrates like sugars and starch and they produce hormones that stimulate the pancreas to produce insulin even before blood glucose rises.

- The insulin response in humans is 2-3 times higher for glucose given orally compared to intravenously.

- This sytesm has been shown to be over producing insulin in breeds susceptible to insulin resistance /hyperinsulinaemia . Requires research.

- HI may be present in the absence of tissue insulin resistance.

- HI may be present in the absence of obesity due to genetics.

-

Excess Insulin and signaling IGF-1 Receptors in the Laminae

- Insulin may be signaling Insulin-like Growth Factor receptors and stimulating the same pathways as per skin cancer resulting in altered cell metabolism including cell structure (cytoskeletal) and adhesion to adjacent cells becomes dysfunctional . The activity of the enzyme AMPK responsible for cell structure may be changed.

- dynamics leading to the 2 physical events, stretching of the lamellar epidermis and dysadhesion of the lamellar basal epithelial cell from the underlying basement membrane and dermis and each other and supporting cells (the para basal cells) .

- Chemo blocking agents may be a new pathway to treating laminitis caused by endocrinopathic issues as in EMS and PPID where hyperinsulinemia is present.

- EVJ 49 (2017) 643-654
- Healthy obese horses do exist.

- Insulin also controls expansion of blood vessels through the nitrous oxide (NO) and contraction through the endothein -1 systems (ET-1).

- Hyperinsulinaemia leads to dominance of the latter system

Insulin Signaling in The Lamellae of the Hoof

Normally insulin controls regular uptake and metabolism of glucose, and fatty acids, the metabolic pathway.

The lamellar basal epithelial cell (LBEC) is central to hoof structure but does not have receptors sensitive to insulin.

Endothelial cells lining the blood vessels do have receptors sensitive to insulin and may be part of the EMS syndrome..

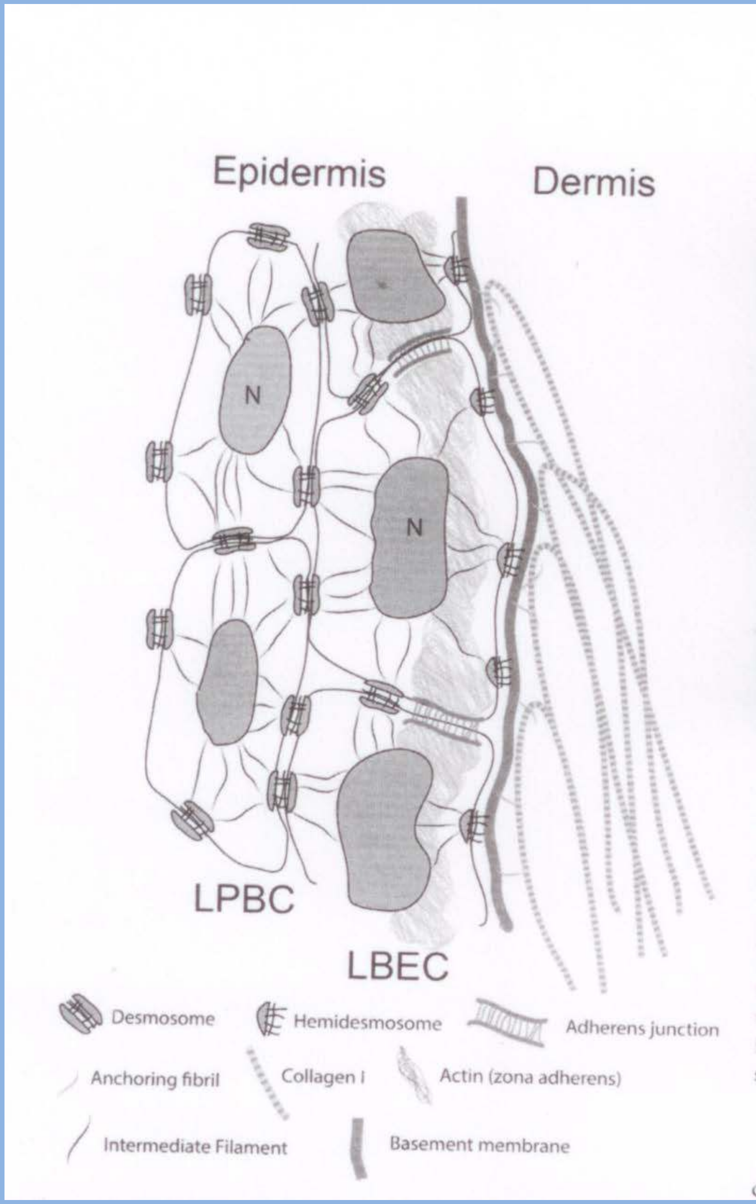
LBEC's do have receptors for IGF-1 sensitive to insulin.

High insulin through IGF-1 switches on the mitogenic (cell division and growth) pathway within cells with the resultant damage .

High insulin through such changes weakens the bond holding the coffin bone to the hoof wall .

Cells can stretch and suffer damage but little inflammation occurs.. Basement membrane separation may not occur to a large extent, it is more stretching and elongation causing damage.

This can be a slow insidius process hard to spot early. The horse may not show pain. It may look like an injury.



High insulin with normal glucose and normal insulin sensitivity Results in laminitis in 48 hours for horses (55 hours for ponies), animals were insulin sensitive and lean and healthy.

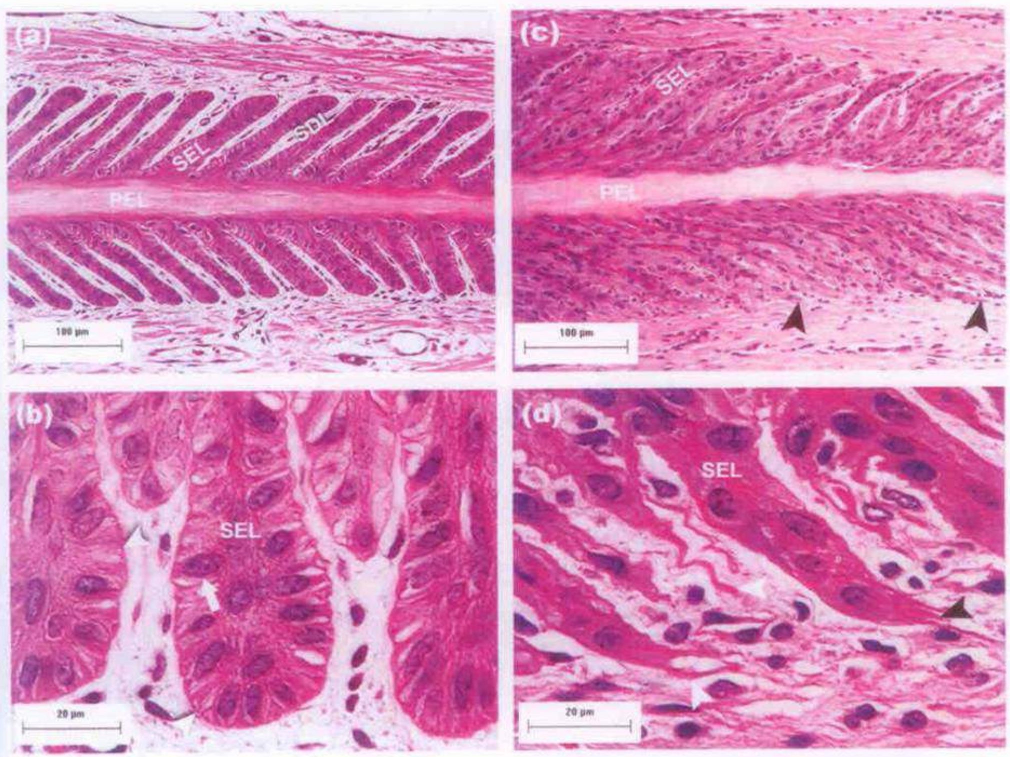


Fig 3: Photomicrographs of hoof lamellar histology from control (a, b) and treated (c, d) horses in transverse section. The secondary epidermal lamellae (SELs) of control horses are uniform in length, show symmetrical angulation to the primary epidermal lamellar (PEL) axis, and have rounded tips. The tips of secondary dermal lamellae (SDLs) extend deeply between adjacent SELs and are close to the PEL. SEL basal cell nuclei (solid white arrow in b) are mostly ovoid and apical, and the lamellar basement membrane is closely apposed to the SEL perimeter (white arrowheads). In treated horses, SELs are lengthened, attenuated with pointed tips (black arrowheads in c and d), and the angulation is asymmetrical. Basal cell nuclei are rounded, and many are pyknotic. SDLs on either side of the PEL are obliterated and many basal cells are confluent. Wavy strands of basement membrane, no longer apposed to basal cells, are present at SEL tips (white arrowheads in d). Stain = haematoxylin and eosin.

Adipose Tissue and Nutritional Obesity due to excess caloric intake

Initially AT serves a protective role by neutralizing free fatty acids and an energy reserve.

High levels of FFA's have been associated with IR (possibly systemic) , liver effects? and disrupted physiology and metabolism in many tissues.

With the accumulation of intra cellular triglycerides adipocytes enlarge to the point where they outgrow their vascular supply, resulting in hypoxia and ultimately necrosis.

The presence of these necrotic cells is associated with leukocyte infiltration .

These leukocytes transform to a proinflammatory state –still unclear, possibly low grade or chronic. They secrete adipokines and cytokines promoting inflammation while decreasing anti inflammatory agents like adiponectin produced by normal adipose tissue.

Adipose tissue in obesity, switches from a normal metabolic state to an abnormal proinflammatory endocrine active state

.

Adipose Tissue and Nutritional Obesity due to excess caloric intake

Composition of the diet can have effects on short term insulin sensitivity even in the absence of expansion of AT.

EG glycemic index for managing EMS.

Not only is basal and stimulated hyperinsulinemia considered a marker/diagnostic criterian for at-risk individuals, **but insulin itself is considered to play direct and indirect roles in the pathophysiology of diseases associated with metabolic syndrome**.

Degree of association of adiposity and systemic IR is non linear.

Breeds /genetics are a factor-GLP-1 (an incretin) is higher in susceptible breeds.

Plasma insulin has been positively associated with the incretin GLP-1 in ponies.

The entero-insular axis stimulates insulin response more in certain breeds.

Degree of IR may correlate well with BW in most cases.

However, not all obese horses are IR

Some relatively lean horses may be profoundly IR-particularly if predisposed breed.

Therefore other factors may disturb insulin signaling and potentially contribute to laminitis besides obesity.

EMS Risk in Arabians

Nature vs. nurture. It's a topic at the center of debate not only in the field of psychology but also when it comes to conditions such as equine metabolic syndrome (EMS). Do horses develop it due to genetics or management practices?

Researchers at the University of Florida (UF) and Cornell University say both play a role in development of EMS, which is characterized by obesity, insulin resistance, and hyperinsulinemia. But they recently linked a spot on a specific gene to EMS—the first genetic locus for EMS to be identified and validated in the horse.

"Our preliminary work seemed to confirm horsemen's lore that some breeds of horse were clearly more susceptible to EMS," said UF researcher Samantha Brooks, PhD.

Arabians, for example, have a reputation for being prone to developing EMS. So, in their study, Brooks and colleagues tested samples from 64 Arabians with histories of laminitis secondary to EMS. They identified markers on *Equus caballus* chromosome 3 near a gene—*FAM174A*—that appeared to be linked with EMS. The team also found that these markers correlated with elevated circulating insulin levels, triglycerides (fats), body condition score, and laminitis—traits associated with EMS diagnosis.

Brooks said this type of genotyping is fairly simple and could be commercially available soon.

"Horses carrying the risk-associated genotype could be monitored more closely from birth and put on preventive diets," she said.

Find more study results at TheHorse.com/39353.

—Casie Bazay, NBCAAM

Owners Recognize Laminitis?

Veterinarians noted more subtle signs of laminitis, such as weight shifting, than did horse owners.

You've heard about the obvious signs of laminitis: the "classic" rocked-back stance, throbbing arterial pulses, and feet that are hot to the touch. But would you be able to recognize more subtle signs of this devastating foot disease in your horse? Researchers revealed that nearly half the owners in a recent study, including those indicating they'd had previous experience with laminitis, did not perceive laminitis signs in horses veterinarians later diagnosed as having the condition.

Researchers evaluated 93 active laminitis cases diagnosed at 25 veterinary practices in Great Britain. Study horses' owners and veterinarians independently completed laminitis reporting forms.

Owners suspected laminitis in 51 horses, all of which were confirmed as having the disease by a veterinarian. Owners did not recognize laminitis signs in the remaining 42 confirmed cases; rather, they cited lameness/stiffness, hoof abscess, or colic.

Common but subtle clinicals signs study veterinarians reported included difficulty turning, weight shifting, and high body condition scores (considered a risk factor). Study owners owners were more likely to report increased hoof temperature, however.

"I would urge all horse owners to educate themselves ... especially about the more subtle but commonly reported clinical signs associated with laminitis," said study author Danica Pollard, BSc, MSc, a PhD candidate at the Animal Health Trust in the U.K.

Read more at TheHorse.com/39428.*—Natalie DeFee Mendik, MA*

Shoe Type Likely Doesn't Change Horse Movement

If you're looking to enhance your horse's gait, you might consider trying a different type of shoe. But recent study results suggest that shoe type won't impact your horse's general biomechanics much.

"The weight of the shoe seems to be what's causing an effect on horses' biomechanics above the fetlock, more than the design of the shoe," said Joëlle Stutz, a PhD candidate working under Antonio Cruz, PhD, of the Swiss Institute of Equine Medicine, at the University of Bern Veterinary School.

They found no remarkable differences in biomechanical movement or hoof structure and function from one shoe to another, Stutz said. However, they did notice a significantly greater front-to-back swing of the limbs with any of the shoes than with no shoes.

"This appears to be an effect of the additional weight

Association exists between markers of EMS (BCS, Plasma trigllycerides and degree of IR) and the incidence of PAL one of most common forms of endocrinopathic laminitis .

The laminitis is insidious in onset and shows a variable degree of lameness.

Insulin is at the center of glucose metabolism and controls not only glucose uptake by insulin sensitive tissues but also cell division and growth and vaso constriction .

AT is transformed in the setting of obesity from a tissue serving as a site of energy storage and production of regulatory molecules to a dysregulated organ which adopts an inflammatory phenotype releasing inflammatory mediators likely leading to a low grade systemic inflammation and possibly changing lamellae tissue metabolism and cell structure.

Markers of inflammation may not be present in EMS

And PPID (Cushing's) disease.

Degree of association of adiposity and systemic IR is non linear.

The old horse

FIGURE 14-3 Example of a thin old horse demonstrating reduction in fat-free body mass

The old horse

FIGURE 14-4 Example of a fat old horse where percentage of fat is greater than fat free body mass.

Adipose tissue-acquired systemic IR-lamellar signaling

- These events in AT disrupt normal insulin signaling in the cells of the peripheral tissues vital to glucose regulation/uptake resulting in IR or ID.

  Due to IR insulin production increases to compensate for the IR present.

- ***Unlike humans, horses beta cell exhaustion rarely occurs, and horses persist in a chronic state of HI (or at least a greatly exaggerated insulin response to a carbohydrate meal), and euglycemia.-compensated IR.

- Lamellar dysregulation /injury is more likely due to effects of insulin on the lamellar epithelial cells (effects not related to carbohydrate substrate availability) .

Adipose tissue-acquired systemic IR-lamellar signaling

- Lamellar inflammation appears to be minimal but activation of IGF-1 receptors in the LEC's and a decrease in an enzyme responsible for cell structure (AMPK activated)concentrations (recent) may disrupt lamellar epithelial dynamics seen in EMS. Pi3 vs mapk systems.

- Other hormone systems may be involved eg increased GH increases IR, leptin (a hormone controling appetite and metabolism) resistance occurs, adiponectin (a cytokine produced by adipose tissue loses anti-inflammatory effects, enteroinsular effects occur.

HI itself may be the main problem in EMS , as the lamellar tissue is affected in a negative manner without obvious inflammatory signs. Lamellar stretching but minimal lamellar separation early in EMS (and other endocrine/hormonal systems like Cushing's or cortisol injections))

However, Chronic hypercortisolemia resulting from primary adrenocortical neoplasia is extremely rare in horses. 20-30%% , but cortisol does have a role.

Insulin possibly an independent risk factor for endocrinopathic laminitis including EMS and PPID.

Inhibited inflammatory responsiveness. Cortisone is anti-inflammatory and may mask signs of inflammation. Beta endorphins may mask the pain making detection difficult.

Laminitis may be the first clinical sign ?

Also non healing /recurring corneal ulcers due to age and PPID Immunosenescense and inhibited inflammatory response occurs with age.

Increased risk of infectious disease possibly subclinical not recognized.

PPID-Traditionally cortocosteroids the cause due to antagonist effects on insulin causing IR. (From Stress or PPID).

PPID

Endocrinopathic laminitis does not develop in a significant number of PPID horses and ponies unless Insulin Dysfunction is a comorbidity. Subclinical laminitis may be present if insulin is high but is not present in PPID animals with normal insulin.

Recent research indicates hyperinsulinemia may be a consistent or at least common finding in animals with concurrent PPID and laminitis.

However whether PPID and hyperinsulinemia have a causal inter-relationship or not, it may be only hyper insulinemia that is associated with lamellar problems.

Hyperinsulinemia and Insulin Resistance may be independent. The prevalence of PPID in older horses and ponies >15 yo diagnosed using ACTH shown to be 21%.

PPID

Hyper insulinemia has been shown to be 27% after excluding PPID in ponies over 15 with age being a significant risk factor.

The probability of hyper insulinemia and PPID occurring (by chance) together in the same animal may be relatively high especially in horses aged 15 and older.

Laminitis in PPID may be a consequence of pre-existing /coexisting EMS or worsening ID. PPID and EMS simply occur concurrently without a causal association in many animals, with both being very common age related diseases.

PPID are at greater risk of Laminitis from any cause, and PPID may be a modifying or exacerbating factor for equids genetically prone to EMS with the result being endocrinopathic laminitis.

Body condition score also was higher in ponies with PPID and laminitis . Key is control insulin for PPID.

The horse as athlete

High maximal aerobic capacity. (VO2 max)

Large intramuscular stores of glycogen

High respiratory capacity of skeletal muscle

Splenic contraction, results in the oxygen carrying capacity of blood increasing by up to 50% soon after the onset of exercise.

Highly efficient and adaptable gait.

Well developed capacity for thermoregulation.

Energy Generation-

Muscular movement requires the transformation of chemical energy stored in substrates or fuels to the kinetic energy of muscular contraction.

All pathways integral to energy supply are concerned with the ultimate production of ATP the final carrier of energy utilized by muscle for contraction.

Energy is generated by the aerobic system utilizing oxygen to burn glucose and to burn fatty acids , or by the anaerobic system which does not use oxygen, and uses glucose with chemical reactions to supply energy.

Fatty acids can provide most of the energy for rest and low level work but are slow release energy sources. HR determines fuels used.

Energy Generation-

s.

CO_2 and water are the end products of aerobic metabolism. More carbohydrate is used as work increases, and fat use stops at higher levels of work.

Excess glucose is changed into fat and glycogen and stored in liver and muscle . Fat not used for energy is changed into triglycerides and stored in adipose tissue and liver with small amounts in muscle tissue.

Once energy needs accelerate and using fats is too slow and oxygen cannot be supplied to burn glucose /glycogen fast enough aerobically , the muscle burns glucose through a short quick method called anaerobic glycolysis metabolism . This is faster but less efficient than the aerobic system, and the end product is lactic acid.

Muscle LA production and eventually lower glycogen and blood glucose contribute to fatigue.

HR determines fuels used.

The horse is designed for speed and power

FIGURE 2-1 The four main athletic species showing the relative maximum speeds during exercise. Maximum speeds in the different species are 19 meters per second (m/s) (Thoroughbred horse), 16.6 m/s (greyhound), and 10 to 11 m/s (human athlete and racing camel).

The horse is superb athlete

TABLE 2–1

Comparative Table for $\dot{V}O_{2max}$, Heart Rate, Peak Blood Lactate, Hematocrit, Stroke Index, and Muscle Fiber Composition

	Human Athlete[a]	Thoroughbred Racehorse	Greyhound Dog[b]	Racing Camel[c]
$\dot{V}O_{2max}$ (mL O_2/kg/min)	69–85	160[d]	100	51
Resting HR (beats/min)	40–60	20-30[e]	100	33
Max exercise HR (beats/min)	190	240[f]	300	147
Resting stroke index (mL/kg)	1.1–1.4[g]	1.3–2.3[f]	–	–
Max stroke index (mL/kg)	1.5[g]	2.5–2/7[f]	–	–
Resting hematocrit (%)	40–50	32–46	54[h]	33[i]
Max hematocrit (%)	40–50	60–70	64[h]	36[j]
Peak lactate (mmol/L)	15	30[f]	20	12
Muscle fiber composition	Sprinters type II >75%	Sprinters type II[e]>80%	Type II >75%	Type I >70 %
	Endurance type I >75%	Endurance type I[k]~30%	–	–

Data from [a]Noakes; [b]Snow; [c]Rose and colleagues; [d]Rose and colleagues; [e]Snow and Vogel; [f]Physick-Sheard; [g]Ganong; [h]Snow and colleagues; [i]Evans and colleagues; [j]McMiken; [k]Rose

Anerobic threshold

Arobic metabolism Can be sustained over long periods of time . The endurance horse. Fatigue is usually due to loss of electrolytes and dehydration and thermal effects , gut issues and mainly lameness issues.

As work intensity and duration increases “anerobic metabolism “ which does not use oxygenecomes more important.

The end products being lactic acid which creates metabolic acidosis

Fatigue is usulally due to increases in muscle LA and decreased blood sugar and muscle glycogen reserves.

.

This creates acidosis.

Anaerobic metabolism cannot be sustained very long.

Even fairly heavy work still uses some aerobic energy such as race horses who use about 60-70% aerobic energy.

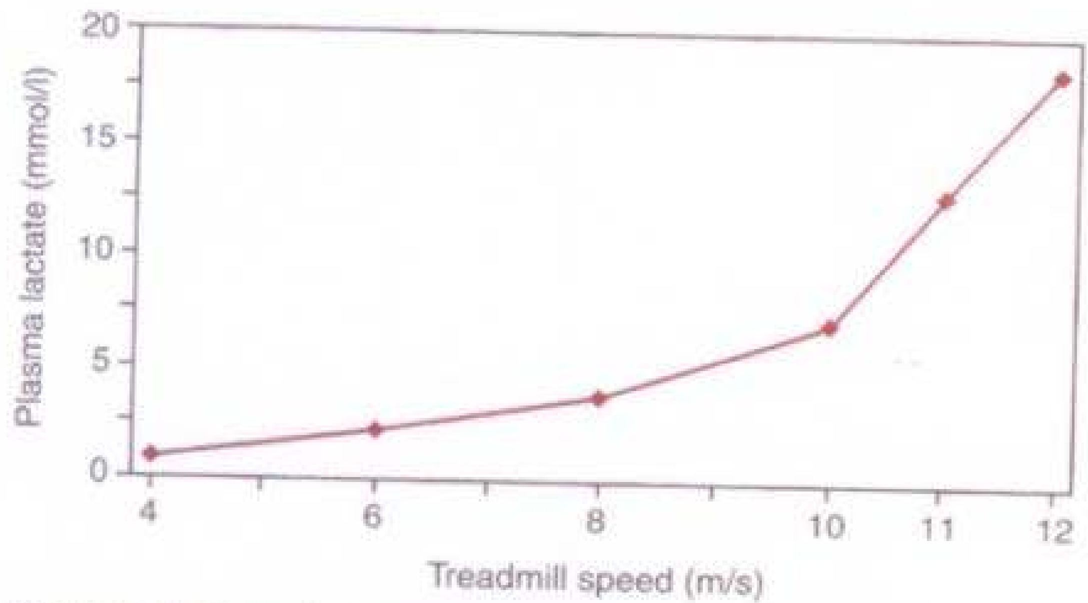


FIGURE 1-5 Normal plasma lactate response to exercise in a 3-year-old thoroughbred horse during an incremental exercise test on a treadmill inclined at a slope of 6 degrees (10%).

Most horses use a combination of aerobic /anaerobic metabolism.

Speed work like racing and 3DE cross country will be more anaerobic. HR over 155 BPM means oxygen can no longer supply the needs for more energy and the anerobic system kicks in.

The barrel horse may not have time to use any fat and will be mostly anaerobic.

Show jumping may be a combination of aerobic and anaerobic at higher levels .

The jump is anaerobic and the canter between jumps is aerobic.

Elite show jumping is half way between heavy and very heavy energy requirements as speed is added between jumps so highly anaerobic.

Most horses use a combination of aerobic /anaerobic metabolism.

The dressage horse and the endurance horse are aerobic .

Horses at maintenance are aerobic and use primarily fat as an energy source .

Transporting horses may resemble an endurance ride (Aerobic)so energy costs should be covered to allow for transport.

If stress increases heart rate the horse will switch to anaerobic metabolism and use up muscle glycogen stores and some blood glucose and fatigue earlier if not wash out before the competition.

Training has a major effect on exercise capacity.

Genetics affect muscle fiber types

Aerobic vs anaerobic use

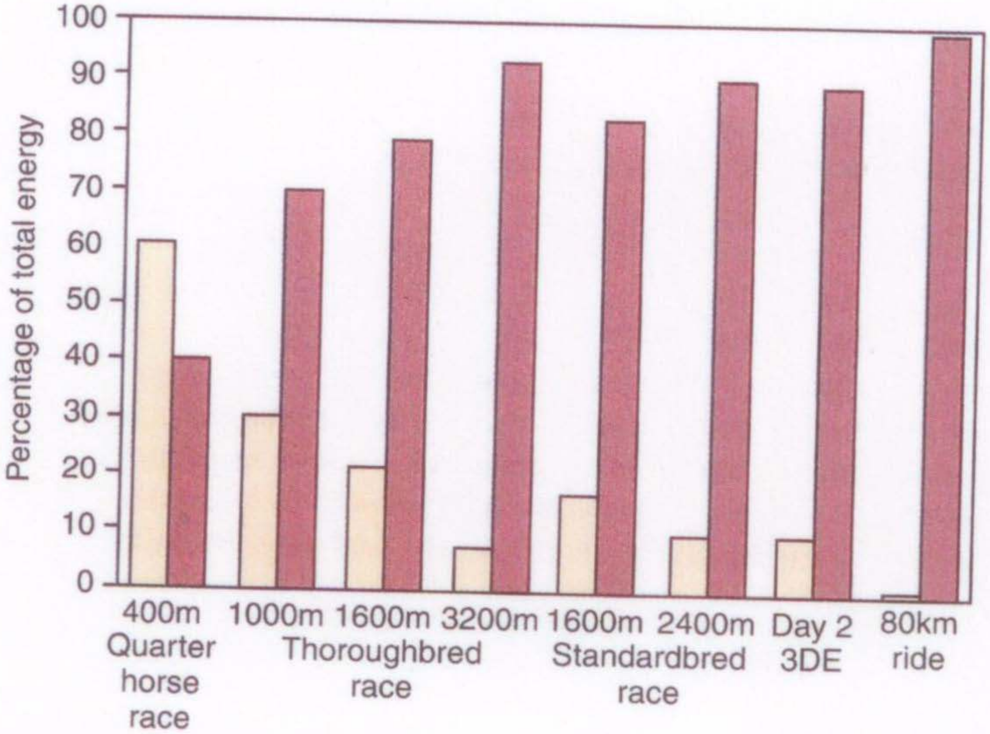


FIGURE 3-9 Energy partitioning. Estimates of the proportion of energy that is derived from aerobic and anaerobic pathways during competitive events (*light shading* = anaerobic contribution; *dark shading* = aerobic contribution).

Fuels used

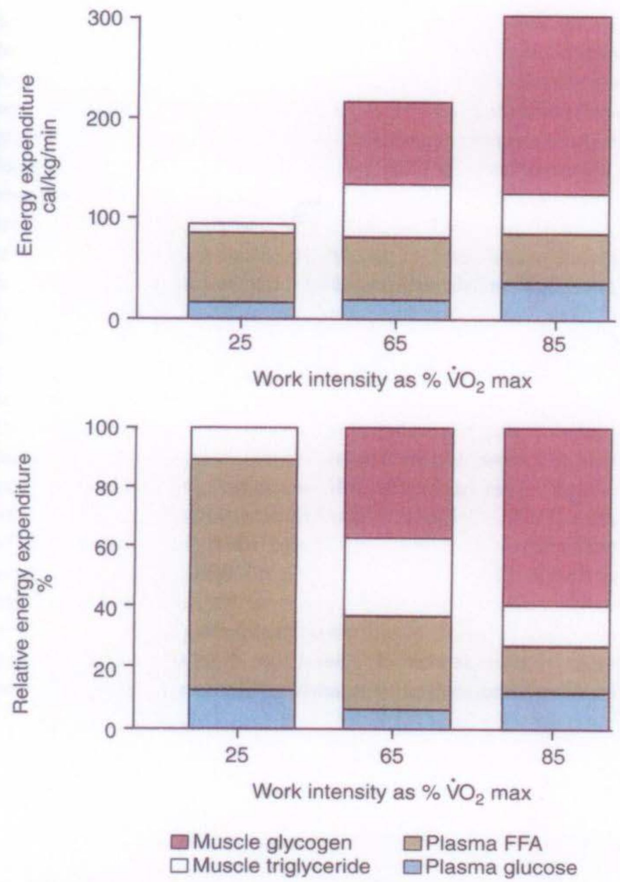


FIGURE 4-1 Total and relative energy expenditure at 25%, 65%, and 85% maximal oxygen capacity ($\dot{V}O_{2max}$) in humans. (*Adapted from Romijn JA, Coyle EF, Sidossis LS et al: Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity and duration,* Am J Physiol Endocrinol Metab *265:E380, 1993.*)

NRC requirement for Energy

500 kg x 0.033 Mcals per kg =16.50 Mcals of energy for maintenance

Light work x 1.20

Moderate work x 1.40

Heavy work x 1.60

Very heavy work =0.036 x BW 1.90

Counting calories!

Special Needs

For easy keepers lower maintenance needs by 10%.

EG Heavy Horses , foundation quarter horses, ponies, Morgans.

For hard keepers increase maintenance needs by 10%.

Eg Thoroughbreds vs heart size .

Senior Horses-teeth stop erupting at 21 years of age .

Minis may need extra care re teeth, and are more prone to impaction colic , and are very efficient and easy to get over weight.

Always maximize forage usage to meet the nutrient needs of horses.

Horses at maintenance can usually meet their energy needs simply by supplying good forage .

E.g. Good grass hay approximately 1.8 Mcals per kg

9.2 kg hay / day can meet the needs of an average horse.

No mold or dust .

Do analysis . Horses eat more alfalfa hay than grass hay. Careful with haylages and clostridium (mouse story) and colic issues.

All the horse may need is a mineral and salt or a ration balancer pellet if protein is needed. Trace minerals are usually low and selenium may be low in this area.

Try for 1.5-2.0% body weight as hay. Never feed less than 1% BW as Hay.

Higher quality hays can be consumed at higher levels so watch intakes. Horses eat more cubed hay. Pasture intakes may be much higher.

Horses do not chew coarser hays more to maximize energy, they eat more up to a point then decrease intakes if very coarse.

Coarse hays may cause diarrhea in older horses and increase ulcers new Danish research.

a

Doing hay analyses

grass hays are modest for calcium and phos and most trace minerals are lower than requirements for even maintenance.

Manganese and zinc are very low on glacial till soils . Copper is low in wet acidic “muck” soils.

Selenium in certain areas is a special case. Usually low (<0.1ppm) especially grey wooded soils but some pockets of higher levels in the interior. Low rain fall tends to keep it in the upper soils layers .

More subject to soil moisture and organic matter of soils.

Because of low rainfall from the sea iodine is also a concern in hays from dry climates.

High rainfall removes calcium and phos in fact creating a reverse ratio where calcium may be lower than phos.

We assume most trace minerals are low in local hays and pastures. Dairy manure will move values up. Potassium may be very high with manure re HYPP.

Iron can be very high in forages grown here or imported.

Energy may be low to quite high.

AND MOST VITAMINS ARE LOW in hays. Do not rely on hays for vitamins and omega three fatty acids . EG -EMND.

Analysis performed by:

Equi-Analytical Laboratories
730 Warren Road
Ithaca, NY 14850
1-877-819-4110
www.equi-analytical.com

Lab Sample No: 21728990
Page 1 of 1

Analyzed for:

Lab Desc: 103
Date Sampled: 07/03/2015
Date Received: 07/13/2015
Date Printed: 07/13/2015
Description 1: RP 1ST CUT
Description 2:
Statement ID: ORCHARD GRASS 2015 - 1st cut

Visit our website www.equi-analytical.com for information on interpreting and using your results.

Results

% Moisture	7.8
% Dry Matter	92.2

	As Sampled		Dry Matter	
Digestible Energy (DE), Mcal/lb		.84		.91
	%	g/lb.	%	g/lb.
Crude Protein	8.7	39.4	> 9.4	42.8
Estimated Lysine	.30	1.4	.33	1.5
Lignin	4.2	19.0	4.5	20.6
Acid Detergent Fiber (ADF)	34.6	157.0	37.5	170.3
Neutral Detergent Fiber (aNDF)	58.8	266.5	63.7	289.0
WSC (Water Sol. Carbs.)	9.3	42.2	> 10.1	45.8
ESC (Simple Sugars)	5.5	25.0	6.0	27.1
Starch	.6	2.6	> .6	2.8
Non Fiber Carb. (NFC)	14.3	64.8	15.5	70.3
Crude Fat	2.6	11.8	2.8	12.8
Ash	7.9	35.7	8.5	38.7
	%	g/lb.	%	g/lb.
Calcium	.35	1.58	.38	1.71
Phosphorus	.19	.88	.21	.95
Magnesium	.18	.82	.20	.89
Potassium	1.37	6.21	1.48	6.73
Sodium	.207	.939	.224	1.018
	ppm	mg/lb.	ppm	mg/lb.
Iron	60	27	66	30
Zinc	19	8	20	9
Copper	6	3	6	3
Manganese	108	49	117	53
Molybdenum	.4	2	.5	.2

	As Fed	100% Dry
RFV		87

Hay Analysis

Moisture- hay over 15% moisture may be subject to mold .

Horses cannot tolerate mold.

Look at protein –level of CP and lysine an amino acid which is a measure of protein quality.

Horse DE measures actual energy content as Mcals/kg or lb.

Other numbers to consider :

ESC =all true sugars

Starch plus ESC directly affect insulin. Keep below 10% DM ?

New research says consider the fructan fraction and the biome, LA production and overall yield of insulin.

WSC= All sugars including grass fructans (compound fructose sugar units) .

NSC =WSC plus starch Keep below 10-12% for metabolic or endocrine issues .

Table 2. Mean areas under the blood glucose response curve (AUC), standard errors (SE) and standard deviations (SD) for all feeds

Mean Areas under Curves (AUC) and Mean Glycemic Indices (GI), Standard Errors (SE), and Standard Deviations (SD) for Means for All 10 Feeds

Feed	AUC	AUC SE	AUC SD	GI	GI SE	GI SD
Sweet feed	28.71[a]	6.4	15.68	128.52[a]	31.87	78.07
Corn	24.73[a]	3.59	8.79	112.60[ab]	13.07	32.01
Jockey oats	19.93[ab]	4.86	11.9	104.83[ab]	24.33	59.59
Oats	27.01[ab]	5.2	18.01	100[ab]	0	0
Barley	16.26[abc]	3.39	8.31	81.15[abc]	22.85	55.98
Wheat bran	15.45[abc]	3.19	7.8	62.93[bcd]	10.44	25.58
Beet pulp	6.45[bc]	2.88	7.01	24.35[cd]	8.35	20.45
Alfalfa	4.18[bc]	0.79	2.75	20.78[cd]	4.62	16.01
Rice bran	4.43[c]	1.74	4.26	13.48[d]	3.39	8.3
Soybean hulls	1.67[c]	0.84	2.05	7.23[d]	3.97	9.71

[a,b,c,d] Values with common superscripts are not different ($P < .05$).

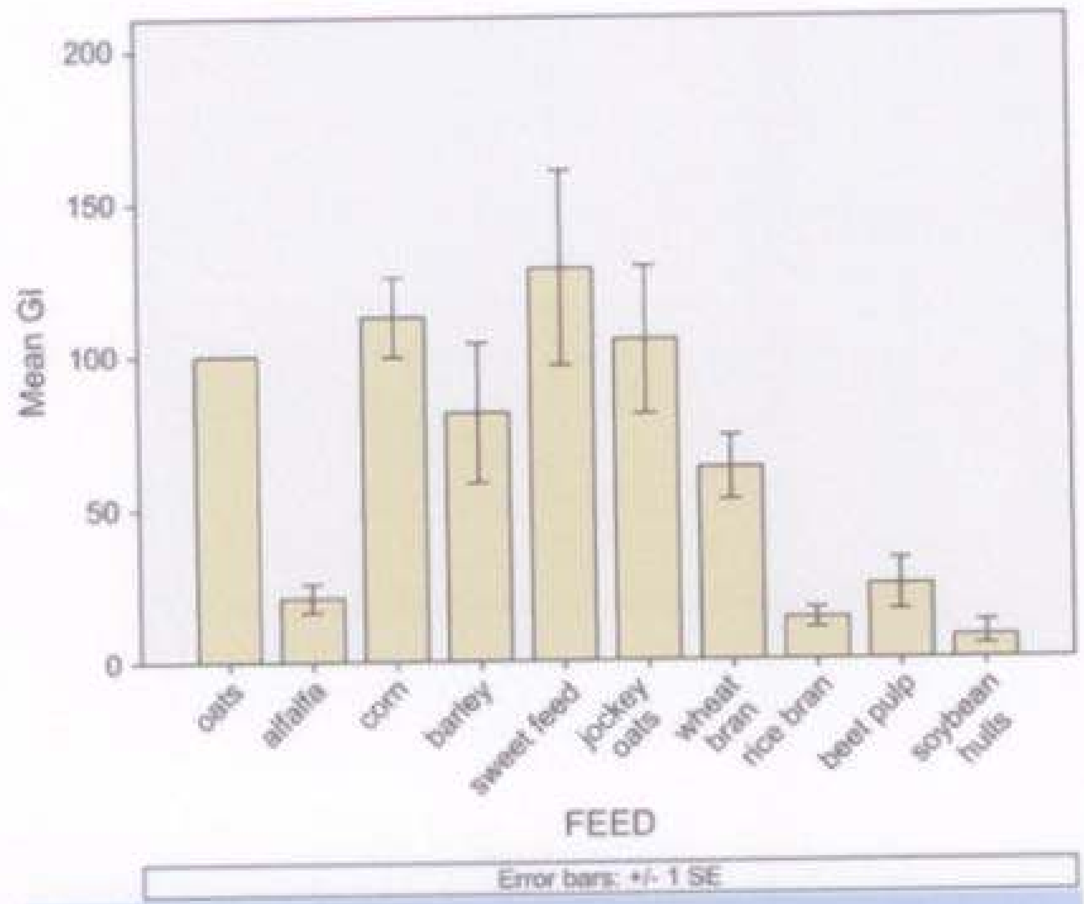
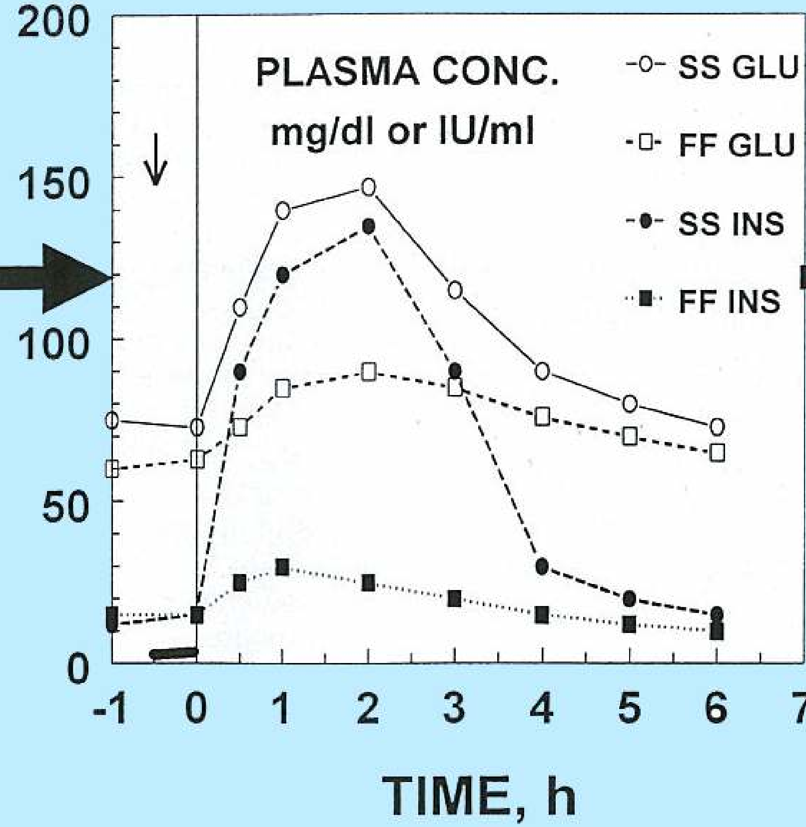


Figure 4. Mean glycemic indices of 10 horse feeds.

ENTRY RATES

Consumption
Gastric emptying
Digestion
Absorption

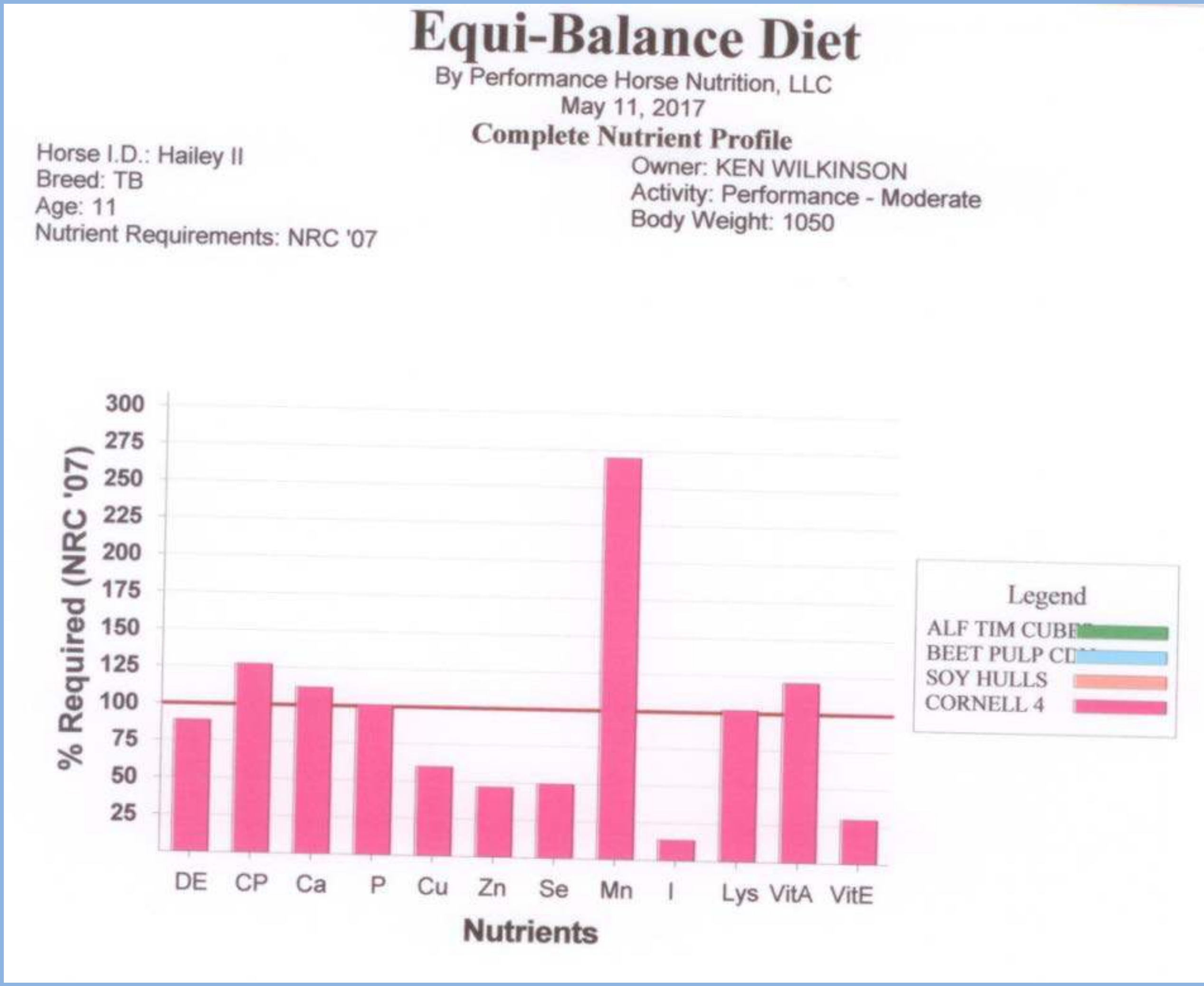


REMOVAL RATES

Glucose-mediated
Insulin-mediated

Maximize Hay

- Evaluate how hay meets NRC requirements fed at approximately 2% body weight or predicted intakes.
- Predicted intakes are 23.6 lbs per day in example.



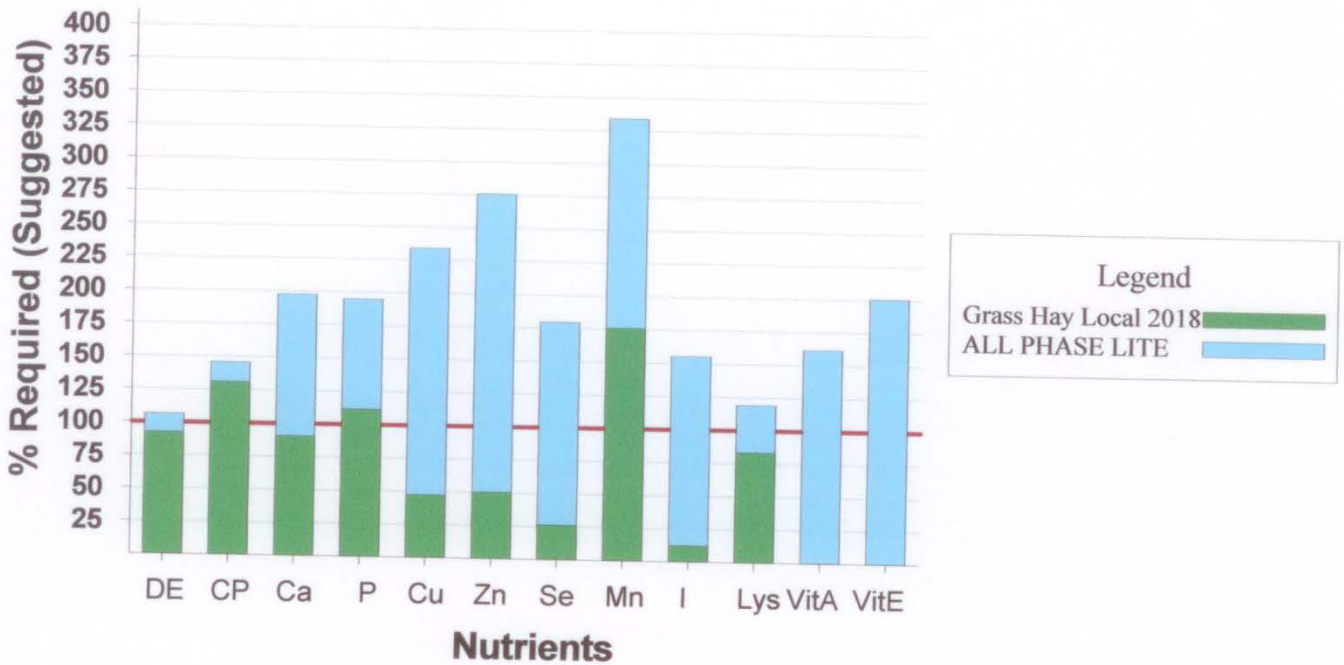
Equi-Balance Diet

By Performance Horse Nutrition, LLC
October 10, 2017

Complete Nutrient Profile

Horse I.D.: hailey maintenance and all p lite
Breed: TB
Age: 12
Nutrient Requirements: Suggested

Owner: KEN WILKINSON
Activity: Maintenance - Average
Body Weight: 1100



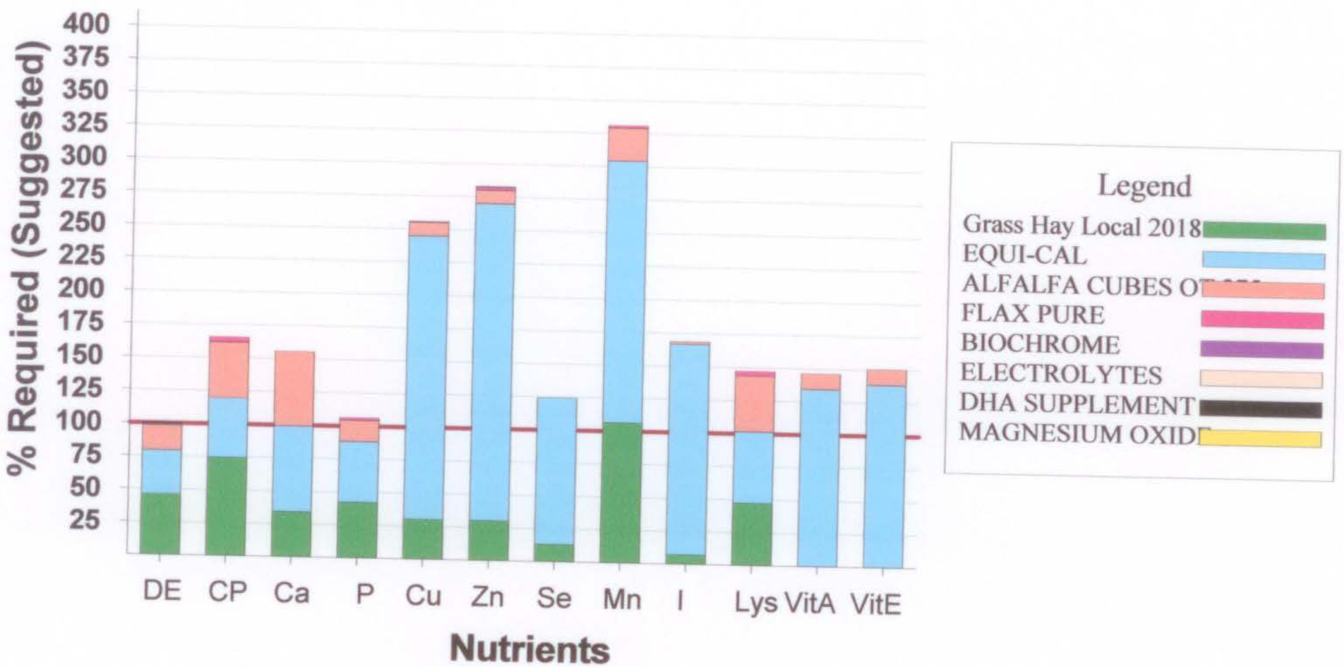
Equi-Balance Diet

By Performance Horse Nutrition, LLC
October 10, 2017

Complete Nutrient Profile

Horse I.D.: Hailey mod work equi cal
Breed: TB
Age: 12
Nutrient Requirements: Suggested

Owner: KEN WILKINSON
Activity: Performance - Moderate
Body Weight: 1100



Horses differ in glycogen metabolism

Glycogen stores are much higher in horses but

Glycogen replenishment following exercise is much slower.

Venous infusion will help

It may require 72 hours for full recovery

Electrolyte supplementation along with hay and grain feeding results in greater glycogen replenishment than does feeding without electrolytes.

Glycogen is stored in a hydrated state. A dehydrated horse is slow to reload.

Differences due to greater initial storage and horses do not increase insulin sensitivity following exercise as do other species, nor increase glycogen synthase as much as other species. They are less efficient at digesting starch and absorbing glucose.

Trace Minerals in the Okanagan

Table 2. Means* and ranges of trace element levels (ppm dry weight)

	Species					
	Timber milk vetch	Arnica	Pinegrass	Kentucky bluegrass	Wheatgrass	Lupine
Cu, $\bar{\chi} \pm$ S.D.,	6.1 ± 1.5	6.4 ± 1.5	7.2 ± 1.7	9.9 ± 2.6	6.2	8.2
Range	2.8 – 9.0	4.5 – 8.5	4.5 – 10.2	7.9 – 14.1	3.9 – 7.9	7.9 – 8.5
Mo, $\bar{\chi} \pm$ S.D.,	$<1.0 \pm 0.7$	$<1.8 \pm 1.2$	$<1.7 \pm 1.5$	5.0 ± 4.0	<1.6	5.4
Range	<0.6 – 3.0	<0.6 – 3.0	<0.6 – 6.0	2.4 – 12.0	<0.6 – 2.4	4.8 – 6.0
Pb, $\bar{\chi} \pm$ S.D.,	1.6 ± 0.4	$<1.8 \pm 0.6$	$<1.9 \pm 0.6$	$<2.0 \pm 0.9$	2.4	1.5
Range	<1.2 – 2.4	<1.2 – 3.0	<1.2 – 3.0	<1.2 – 3.6	1.8 – 3.6	1.2 – 1.8
Zn, $\bar{\chi} \pm$ S.D.,	20.8 ± 6.4	23.3 ± 10.4	22.7 ± 5.6	16.6 ± 2.9	19.8	25.4
Range	9.2 – 38.6	13.0 – 44.5	12.5 – 33.4	13.8 – 20.7	19.0 – 20.7	14.3 – 36.5
Se, $\bar{\chi} \pm$ S.D., Range	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Number of samples	36	8	15	5	3	2

*Mean ($\bar{X}$) calculated using minimum detectable level.

Selenium levels Okanagan

Performing your original search, ***Anywhere in Content: forages in south okanagan***, in NRC Research Press will retrieve 25 citations

Article

« Previous TOC Next »

SELENIUM CONCENTRATIONS IN BRITISH COLUMBIA FORAGES, GRAINS, AND PROCESSED FEEDS

PDF (659 K)

PDF-Plus (313 K)

Citing articles

J. E. MILTIMORE, A. L. van RYSWYK, F. M. CHAPMAN, C. M. KALNIN, W. L. PRINGLE

Canadian Journal of Animal Science, 1975, 55(1): 101-111, 10.4141/cjas75-013

ABSTRACT

Samples of hay, silage and grains grown throughout British Columbia and samples of ration components available in British Columbia were analyzed for selenium in order to characterize the selenium status of feeds available for livestock. There were nutritionally important differences between the same feeds grown in different regions and between different feeds within a region. Even within a region featuring relatively high selenium concentrations, many feeds were found to be below the 0.1 ppm minimum requirement for livestock. A selenium analysis of feeds appears necessary in order to supplement livestock appropriately.

Selenium Most of B.C. Island, Okanagan-Central interior

An area generally deficient but local levels may vary., usually less than 0.1 ppm in the Okanagan. Be aware alkaline soils are more conducive to selenium uptake.

A major component of enzyme glutathione peroxidase. Protects cell membranes.

Required for the control of thyroid metabolism. Activates thyroid hormone.

For fertility.

Deficiency more pronounced in foals with WMD at birth.

Mature horses partly protected by other factors but these drop off with age.

recent mortality reported from Washington state U with mature horses showing myo-necrosis of head and skeletal muscles, subcutaneous edema, pleural effusion, and ulcerative glossitis (an ulcerative and inflamed tongue). Suggests a full body involvement. The glossitis probably from pica due to mineral deficiency. Mortality had occurred. Herd wide selenium deficiency and myodegeneration were found. Watch for ventral edema, weakness and death.

The US studies were quickly extended to include neighboring areas in Canada, thanks to work by Lessard et al. (1968), Walker (1971), Winter and Gupta (1979), Miltimore et al. (1975) and a number of others, and this has led to an expanded map (Figure 28).

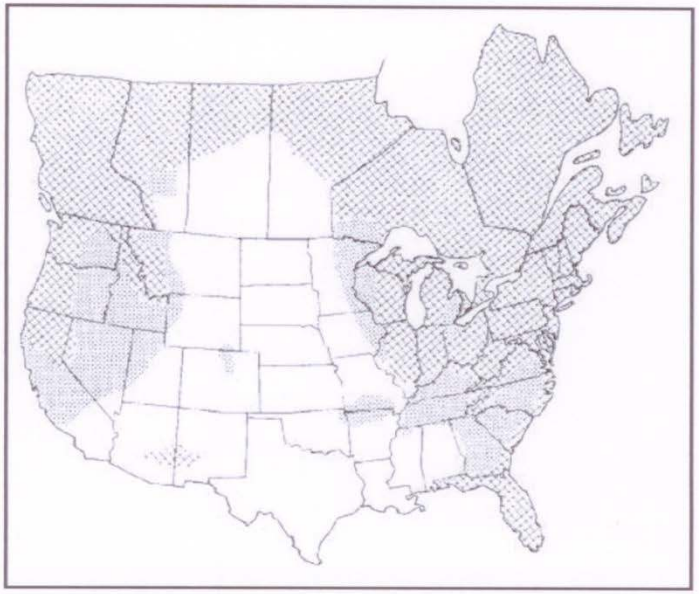


Figure 28. Selenium contents of forages and grains, relative to their adequacy for animal health, in Canada and the United States (after NRC, 1983)

This map is broadly generalized and suggests that in Canada areas of selenium deficiency tend toward the east and west coasts, with the central, prairie provinces being largely adequate. There are many localized variations, however, and persons interested are referred to the literature citations for more exact data.

Selenium Okanagan-Central interior

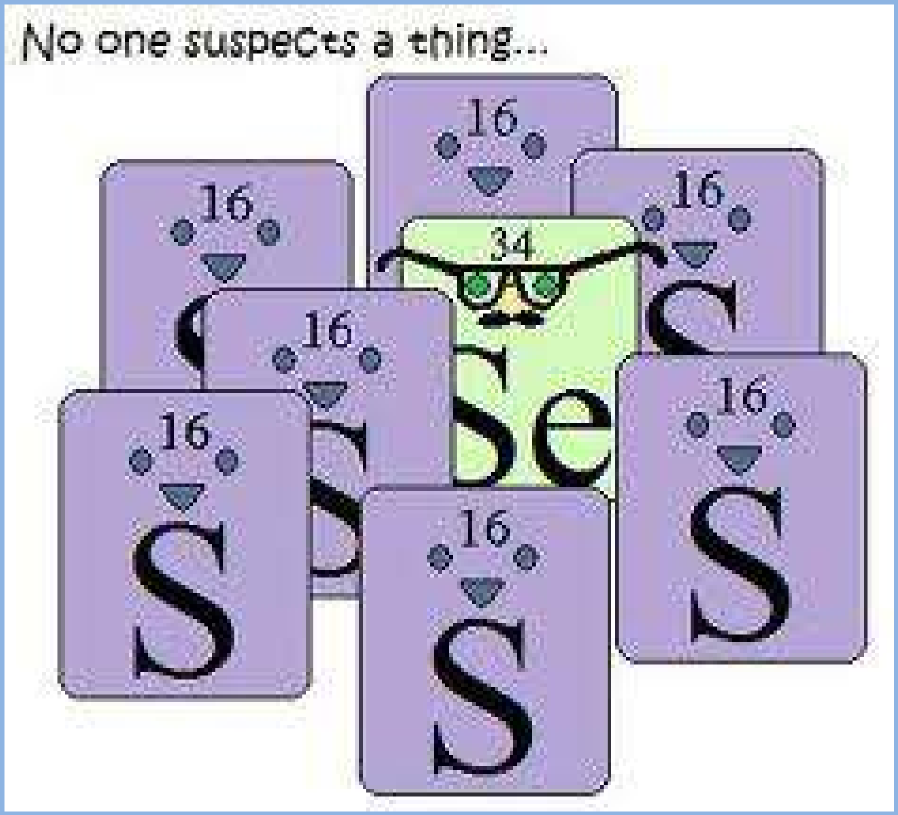
Vitamin E in green grass may mask a borderline deficiency.

Affects keratinization , Acute excess =blind staggers or chronic excess =alkali disease. Foot and coat issues.gut, lung, heart and kidney issues.

Excess Se may replace sulfur in tissues.

May only see poor frog and chronic thrush in marginal excess.

2-3 mg/day recommended no more than 5 mg/day.

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Copper-average values are low in the Okanagan, often less than half NRC

In multiple cellular functions
Mobilization of iron stores and haemoglobin formation.

Myelination of nerves

Keratin formation eg for cracked feet in the Okanagan ,

Part of the enzyme controlling connective tissue formation in the fetus and growing horse drives the formation of cross linkages in collagen fibers. The mare must provide needs for the foal as milk is low and foals rely on reserves when born.

For bone formation. Ensure the pregnant mare is well supplied as the foal depends on reserves when born.

For melanin synthesis ie coat colour.

Other trace minerals may interfere with copper absorption. Use 10 or 5 -1-3-3 ratios for iron, copper, zinc and manganese. Iron levels can be very variable.

Suggest 10-15 ppm for most classes of horse and broodmares respectively. Some suggest 25 ppm for broodmares.

Zinc- along with manganese most glacial soils are low as are plants

Functions in many tissues a component of many enzymes. Highest in pancreatic tissue and hoof horn and liver. Intermediate levels in muscle.

Required for replication of DNA/RNA and cell growth and gene function especially reproduction.

For insulin function.

For cognitive function

Linked to skin health. Deficiency causes parakeratosis. Inappetance in foals, reduced growth rate, alopecia, and decreased horn strength and hardness.

Oversupply may tie up copper causing OCD if extremely high . Ratio of 3:1 copper ideal as they share the same absorptive mechanism.

Manganese-glacial soils are very low in manganese and zinc.

Essential for many enzymes. For carbohydrate and lipid metabolism.

For synthesis of chondroitin sulfate necessary for cartilage formation.

Protects cells from oxidative damage.

Involved with fertility and central nervous system function.

There is no manganese storage in the body as per selenium which has no storage depot. It must be there every day.

Milk levels are low.

Suggested levels are 40 ppm NRC . Relatively safe to use at higher levels.

Ratios as per other trace minerals

iodine

Natural hays ad pastures will be very low . Unstable in heat when curing hays.

Provide 2-3 mg/day

Very difficult to analyze for.

Selenium is required for activation of iodine thyroid hormone.

Easy to over-supplement with some kelp based supplements.

Do not rely on salt block consumption to supply salt and iodine, just a back up.

Pasture grasses-carbohydrate metabolism

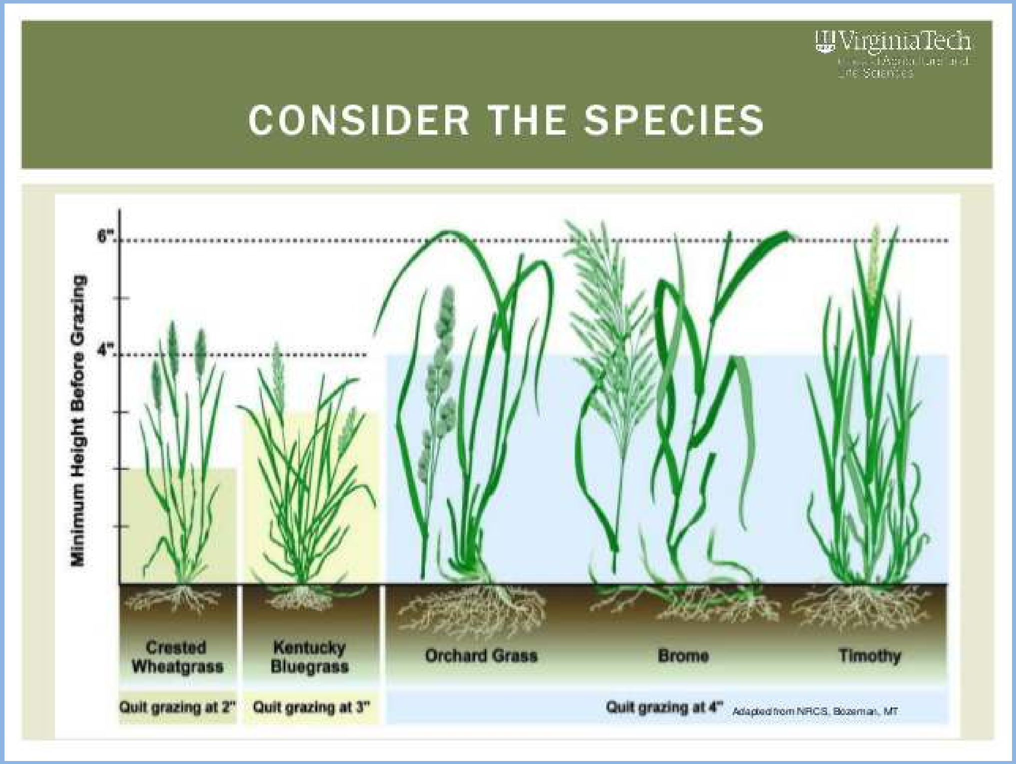
Our grasses are called C3 (cool season) type grasses and they store high levsels of fructans as their main storage carbohydrate. Plus some simple sugars and lower levels of starch.

All carbohydrates can be greatly increased and energy levels increased and potential for PAL may increase depending on growth pattern.

These grasses can do photosynthesis when various stresses shut down growth.

Respiration-grass uses the sugars produced to create energy and raw materials to make other compounds.

The critical low temperature is 5 Centigrade for respiration.



Grasses

Grasses cut as hay may lose 30% of sugars during curing.

Sugar content is affected by-

Shade or Cloudy days

Weeds may be higher in sugars than grass

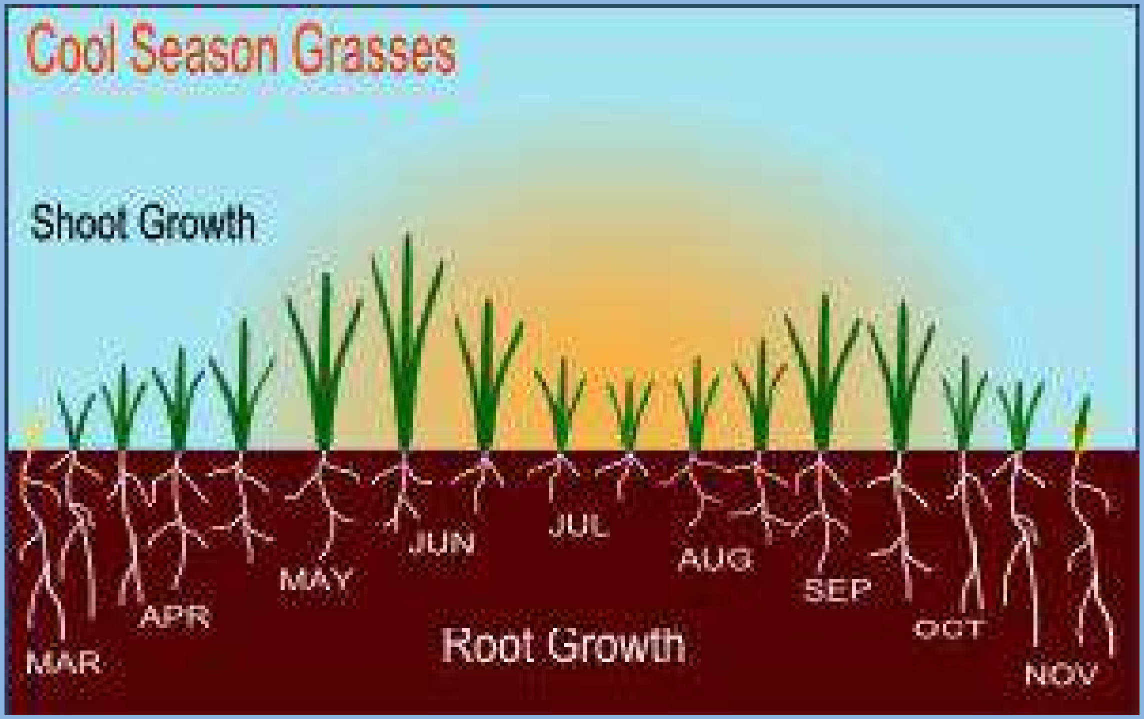
Cool nights and long days may create highest sugar levels

E.g. Laminitis in spring.

Usually lower levels in am max late afternoon.

May double morning to night

15-30% dm as fructans and simple sugars.



Grasses and sugars

Stressed grass is higher in nsc’s

Horses are at more risk for laminitis during times when temperatures fluctuate. The 5C rule.

NSC are high when cooler and lower when warmer but higher if drought stressed.

Hardening. When grass freezes the fructan is broken down to simple sugars which act as anti freeze. Higher sugars improve freezing tolerance.

Green grass recently frozen will be higher in simple sugars as will grass recently drought stressed.

Figure 5-1 Seemingly dead grass, dormant from drought, with stores of fructan or starch in the crown and stem bases (left). After rain, the stores are used up to produce new growth (right) until such time as the grass has enough leaf area to start producing more carbohydrates.

Drought and salinity and fertilizer

When water supplies fail, the grass will compromise enzymes involved with respiration before photosynthesis stops.

The grass can gather reserves of energy before going dormant.

A plant with a generous store of fructans and sugars will be ready to grow when the rains return.

EG 35-40% of orchard grass after a drought. As mostly large fructans .

OG and ryegrass have showed fructans and sugars rose to over 40% during 45 days of drought.

It requires research under your conditions and grasses .

Salinity will increase NSC in grasses.

Lack of nitrogen and other nutrients will increase NSC's

Grasses-heat stress

The optimum temperature is 5-32C for cool season grasses.

When at 30 C the cool season grasses start to get heat stressed.

The start of hot weather often coincides with peak concentrations of fructans and sugars and starch.

Under heat stress C3 grasses may slow photosynthesis and photorespiration increases.

They can lose NSC's as they cook to death.

Figure 16-1 Dry, frosted grass, yellow in appearance, can have a dangerously high sugar and fructan concentration. The grass illustrated is Phalaris photographed in a horse paddock near Canberra.

Myths

Lush grass may not be higher in NSC's

Dry brown stressed grass can be dangerously high in sugars.

Colour has nothing to do with NSC content. Dead brown grass can be high in sugar and cause laminitis.

Proper moisture hay does not decrease in sugar content with storage.

Coarse stemmy hay may or may not be high in NSC. Maturity may vary.

Weeds have no feed value.

9. High NSC Weeds

Certain weeds can be very high in sugar, starch or fructans and some of these are palatable to horses. Forage researchers know that weeds are palatable and high in nutrients, and some of those are already being further improved for cattle forage. Some common weeds that can be high in sugars or fructan include:

Dandelion *(Taraxacum officinale)*

Dandelions are found around the world and are known to be highly palatable to horses (*Figure 9-1*). Dandelion has more fructan than grass (Chatterton, personal communication). It is the same type of fructan (inulin) that induces experimental laminitis when administered to horses (Van Eps and Pollitt, 2006). After long term drought has killed shallow rooted grass, dandelions, with a deep tap root full of reserves, may be the only thing left green in the pasture. The concentration of sugars and fructans may reach 25% DM in above ground parts under conditions of repeated frost and sunny days (Watts, unpublished data) and 12-15% of fresh weight as inulin in the leaves (Van Loo et al., 1995).

Figure 9-1 Dandelion (Taraxacum officinale), a palatable pasture weed may contain up to 25% sugars and fructans as dry matter in its above-ground leaves and stems.

Storksbill or redstem fillaree *(Erodium sp.)*

Additional concerns

The stomach of the horse creates issues.

Small Secretes acid 24/7

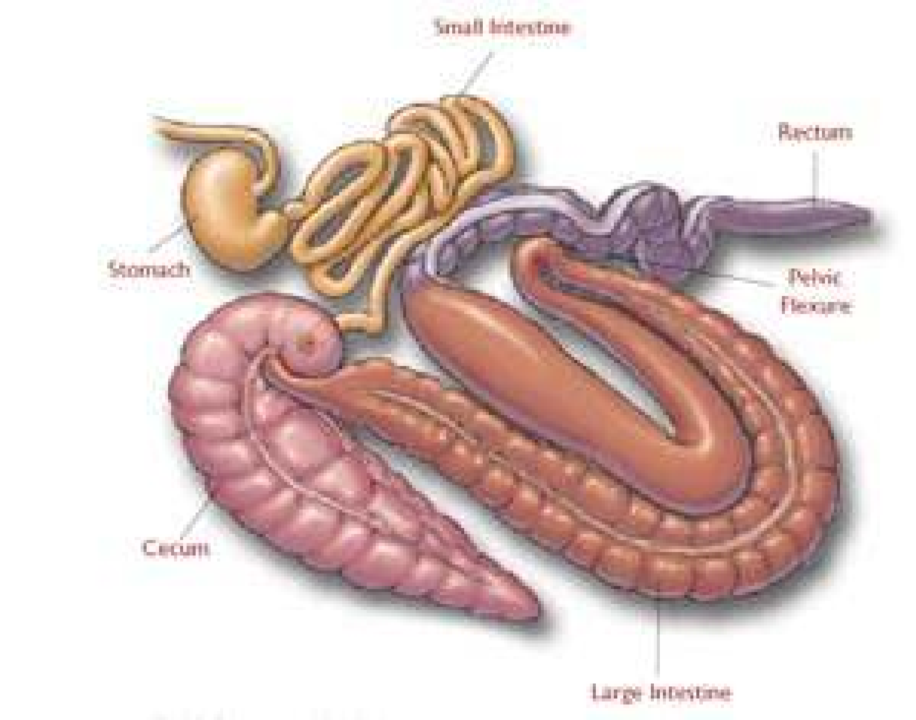
The upper half is not protected

Ulcers are an issue for performance horses, even pleasure horses and wild horses.

Acid production is stimulated by stress hormones or excitement or exercise.

A limited capacity for starch digestion . The small intestine is shorter and does not have the same glucose transporters as other animals

The horse cannot digest fructans



Stress

New research shows 10 grams of magnesium from a good source like citrate or oxide will reduce the fear response by approximately one third.

The stress hormone cortisone may be destructive and may increase insulin levels.

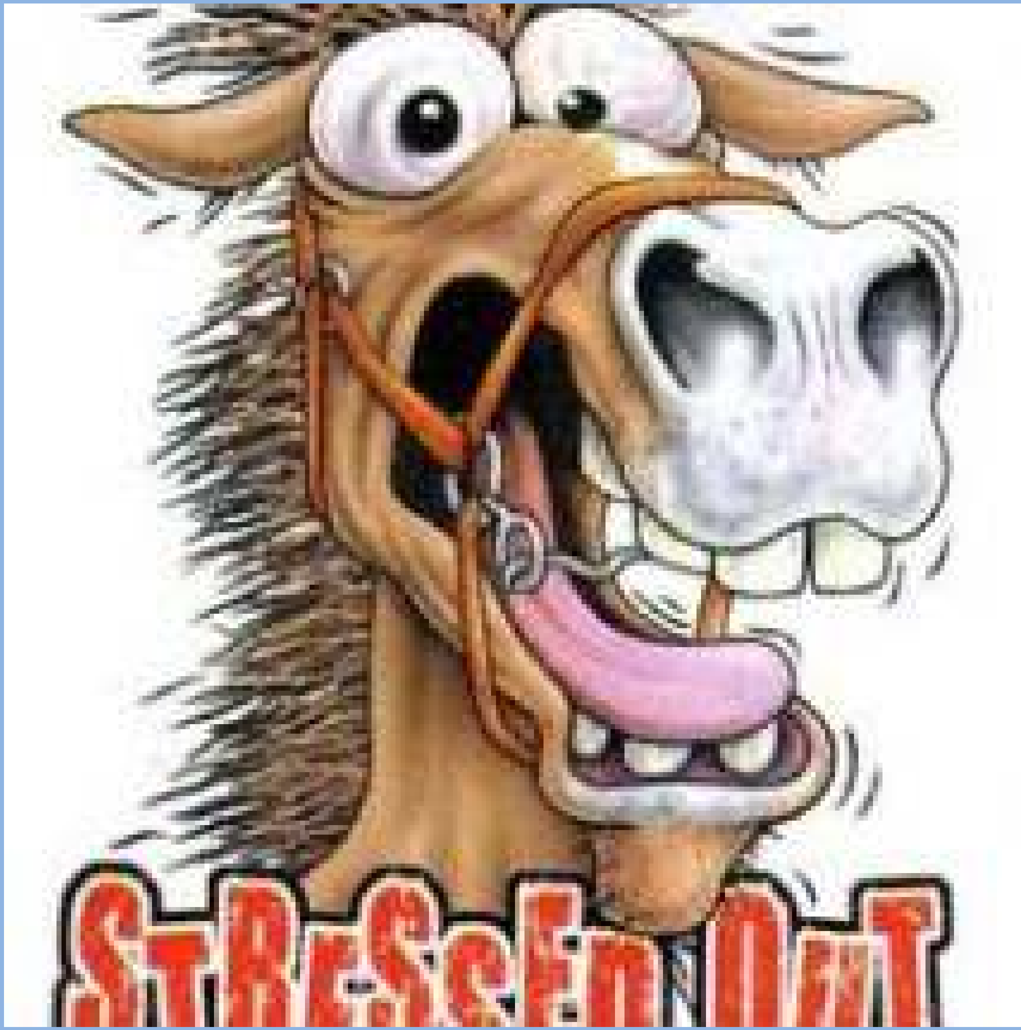
Adding some alfalfa will help buffer the stomach . Time the addition .

Extremely coarse forage may cause ulcers in addition to impaction colic especially for minis.

Feed little and often

Horses do not have the capacity to digest starch as well compared to other animals . Excess starch can easily flow through to the hind gut.

Try not to leave horses without food for longer than 5-6 hours. They must buffer the stomach somehow.



Horses Issues

Horses do not sweat like humans . Ensure electrolytes lost are replaced. Mild saline may enhance hydration recovery. Offer non saline water as well.

Horses do not drink enough in cold weather. Impaction may occur. Top dress if possible. Hydration is difficult to assess.

Water intake is driven by chewing . Maximize forage.

The thirst mechanism is shut down by stress or excitement.

Horses over 20- teeth and chewing become a bigger issue.

Minis may have tooth issues and colic issues.

Feeding Fat and Fiber

- Fat has 2.5 x the calories of regular grains.
- Lower Bulk but slows gastric emptying and reduces glycemic response even with grain feeding.
- Requires less water for utilization
- Lower heating effect from digestion, may lower thermal load e.g. For endurance horses.
- High calories without the risk of rapid fermentation or starch overload low GI
- Lowers dust levels for sensitive horses.
- Burned aerobically only.
- New information shows horses are more aerobic than previously thought.

Other factors- Cold temperatures-

A mature horse

If well fleshed and a good coat with good shelter may have a lower critical temperature of about -15C and require 2.5 % more energy for maintenance per degree C below that.

A baby horse 21 C LCT

Growing horse 10- 0 C LCT

Requires about 1.5% more energy for maintenance for each degree C below the LCT.

Cold, wet and windy conditions can increase energy needs over 50% above normal.

Provide 8-10 inches of bedding in shelters.

Old horses may need special stabling.

Blanketing may help save 10-15% on the feed bill in cold weather

Other additives

Horses not on green grass will benefit from adding some omega three fatty acids.

Normally green grass has a high level but hays have less and they are not stable during storage.

We suggest adding milled flax or DHA type supplements .

Sweat losses

Sweating rates may hit 10-12 liters per hour in extreme situations.

Horses are unique having a hypertonic sweat . The higher salt levels increase heat losses during evaporation. The sweat is high in the protein latharin. This helps the sweat adhere to the body drawing more heat.

The sweat is higher in electrolytes than plasma so the horse sensors read the plasma as being more dilute as sweat losses increase

The horse thinks it is well hydrated and the thirst response is not triggered .

However it is steadily losing fluid to the point where catastrophe may occur.

Note- excitement or fear will shut down the thirst response further aggravating the situation. They also increase acid production.

Fluid and electrolytes

The horse has unique adaptations to shed heat via sweat.

First increases in hydrostatic pressure during exercise enhance fluid shifts from the vascular compartment to the interstitial space increasing the availability of fluid for sweat production.

Second the sweat gland of the horse is very simple and sweat excretion less complex. Unlike the human sweat gland, it does not respond to aldosterone which helps absorption of sodium and thus cannot conserve sodium which in turn attracts water back into the body.

The sweat gland in horses acts like a funnel to allow a slightly hypertonic (concentrated) solution of electrolytes to move from the interstitial space to the surface.

The extra salt and a protein latherin also alter the evaporation point enhancing cooling.

Sweat losses

During submaximal exercise horses under high heat and humidity can lose 12 L per hour.

This large volume of sweat results in proportional decreases in body weight , total body water and plasma volume.

This can compromise venous return , cardiac filling pressure, cardiac output, and the ability to thermo regulate.

Blood flow is then reduced to the non obligate tissues to maintain flow to the muscles etc. This may compromise gut health causing colic or affect kidney function. Heart rate must go up to compensate.

Thermal regulation is sacrificed for muscle supply . Shock may occur.

Water and Hot Weather

Horses can lose 2-4 gallons of sweat per hour. Horses normally drink 5-10 gallons per day.

Less evaporation occurs during high humidity.

Thermal neutral zone is about 5C-25C

Heat stress reduces feed intakes. Less feed especially forage means less water intake and less chewing and buffer added.

Horses can adapt after 14-21 days to heat and humidity.

Provide clean fresh water at 45-64F temp. At all times.

Water intakes may double in hot weather. Use top dress electrolytes 4 hours before work and consider pastes or 1 oz every 1 hour or 10 miles according to vets advice (add on feed at rest stops with water).

Use electrolyte water and plain water free choice to rehydrate.

Transport in coolest part of day water every 4-8 hours.

summary

Idle horses drink about 25 liters per day

Range 20.5-33.5 l/d

Drink more water with all hay diet 3.2-4.4:1

Vs grain mix 2-2.60:1

More grain means less water consumed.

Lactating mares need at least 37.5-50 liters or more depending on diet, temperature, possibly 40-70 l per day.

Suckling foals need water and respond best to a bowl or bucket easily accessible at 75-90 cm high.

Horses fed dry feed drink around time of feeding and prefer cool to luke warm water. They only drink 5-6 minutes /day. Possibly 5-8 gallons per day at maintenance. Horses drink less cold water, 20C is ideal. Colic may be an issue if water is cold in winter.

If feed deprived, they spend more time drinking.