



Wildwood Farm CLIPS & CLOPS Oak Harbor

SEPTEMBER 2025

YOUR NEIGH-BORHOOD HULLABALOO

CREATED & EDITED BY HEATHER CARDER

The Evolutionary Mistake That Made Horses Into World-Class Athletes

Story by StudyFinds

In a nutshell:

A genetic mutation that should have broken a critical protein in horses instead gets cleverly “recoded,” allowing their cells to produce more energy and better resist stress during intense exercise.

This mutation, shared by all living horses, zebras, and donkeys, likely evolved in a common ancestor around 4–4.5 million years ago and helps explain their extraordinary oxygen consumption and athletic endurance.

The discovery challenges assumptions about how genes work in mammals and could inform new treatments for human diseases caused by similar stop codon mutations, which affect about 11% of genetic disorders.

NASHVILLE — Thoroughbred racehorses are biological marvels that consume oxygen at astonishing rates, more than double what elite human athletes can manage. During intense exercise, these animals process up to 360 liters of oxygen per minute, feeding the enormous energy needs of muscles that make up over half their body weight.

For years, researchers have wondered how horses evolved this exceptional capacity. The fossil record tells us they started as dog-sized leaf browsers before transforming into the powerful runners we know today. But the molecular changes that enabled this transformation remained hidden, until now.

The Genetic Puzzle of Horse Power

A team of researchers from Vanderbilt and Johns Hopkins recently uncovered a fascinating evolutionary twist that helps explain horses’ metabolic superpower. In work published

in *Science*, they found what initially looked like a genetic error that should have been harmful but instead became an advantage through clever biological trickery that boosts energy production.

The researchers examined a system that controls how cells handle stress from burning oxygen. This system becomes especially important during exercise. It involves two main players: a protein called KEAP1 that acts as a sensor for stress, and another called NRF2 that turns on protective genes when needed.

When looking at the KEAP1 gene in horses, zebras, and donkeys, the scientists spotted an odd genetic “stop sign” early in the gene’s instructions. Normally, this kind of mutation would prematurely end protein production, creating shortened, non-working proteins that cause disease. Yet these animals not only survive with what should be a harmful mutation, they excel as some of nature’s greatest athletes. This contradiction made

Continued on page 11.

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- Ergonomic grip designed for equine comfort
- Precision head shapes targeting muscle tension
- quiet motors that won't spook
- Packaging designed by us, because we know what works.



- Super comfortable for horse's ears. It is below the 40db startle threshold for spook-prone breeds.
- 8-head muscle targeting simultaneously treats paired muscle groups: Hindquarters and shoulders. 20mm reach accesses deep fascia layers where 68% of equine tension resides.
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- 9 speed equine presets: Competition mode, Recovery mode, Chronic Pain mode.
- Farrier-approved ergonomics: 45-degree angled neck for hard-to-reach stifles and gaskins; non-slip grip even when using oils; weighs 2.2 lbs, light enough for grooms to use all day.

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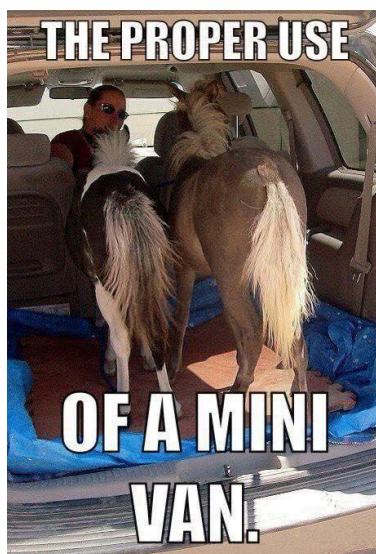
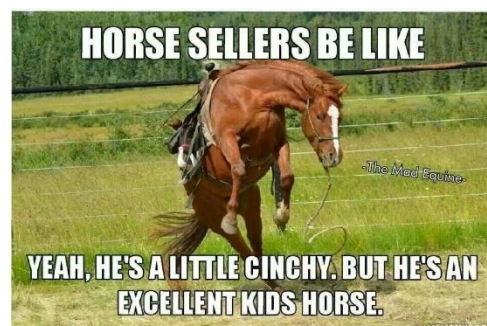
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New Items for September

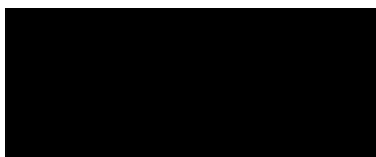
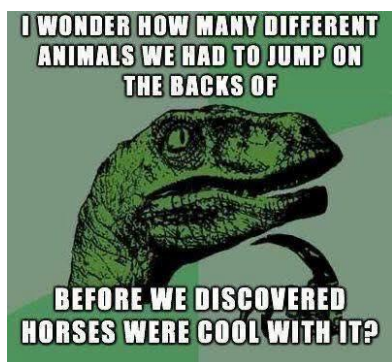




Jumping lessons:
when your horse suddenly does sliding stops
better than a professional reining horse



"Don't tickle the horsey till Daddy comes out from behind ... never mind."



**That moment when
You make eye contact
With the judge:**



My face:



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**PACIFIC NORTHWEST
RIDING ACADEMY**



August 1, 2025

Summary for 07/01/2025 - 07/31/2025

32 alerts posted reporting on 48 confirmed cases

Ordered by State:

Disease	State	Confirmed
Eastern Equine Encephalitis	Florida	2
	Louisiana	11
	Ontario	1
	South Carolina	2
Equine Herpesvirus- Respiratory	Washington	1
Equine Infectious Anemia	British Columbia	1
	Kansas	5
Equine Influenza	Connecticut	1
	Wyoming	2
Pigeon Fever	Washington	1
Potomac Horse Fever	Pennsylvania	1
	Virginia	1
	Washington	1
Rabies	Kentucky	1
Salmonellosis	Maryland	2
Strangles	Florida	1
	Michigan	3
	Ontario	5
	Washington	1
	West Virginia	1
West Nile Virus	Colorado	1
	Kansas	1
	Kentucky	1
	Virginia	1

Equine Disease Communication Center
 4033 Iron Works Parkway
 Lexington, Kentucky 40511
 Phone: 859-233-3322
 Email: edcc@aaep.org

Nutrition Corner

5 COMMON HORSE FEEDING MISTAKES

- 1. FEEDING BY SCOOP OR FLAKE INSTEAD OF BY WEIGHT.** Each concentrate of complete feed, while similar in volume, differs in weight. With regards to hay, grass hays weigh different than alfalfa and even a 2 lb. difference in feeding can affect microbes in the gut.
- 2. FEEDING SENIOR FEED UNNECESSARILY** A common mistake horse owners make is feeding senior feed to a horse that does not yet have a need for it. Senior feed is designed to be a complete feed. You might be doing your horse a disservice by not meeting their micronutrient requirements.
- 3. OVERESTIMATING HOW MUCH WORK YOUR HORSE DOES** Considering that domestic horses evolved from wild predecessors who traveled 20-30 miles per day to meet their feed and water needs,

It is clear that most domestic horses are not getting as much exercise as they used to. We tend to overestimate the amount of exercise they DO get.

- 4. OVERSUPPLEMENTING OR CHANGING SUPPLEMENTS FREQUENTLY** A bunch of supplements that were not meant to be fed together can create competitive inhibition and interactions between macro and micronutrients in the horse's gastrointestinal tract.
- 5. FEEDING IN MEALS INSTEAD OF CONTINUALLY** Horse's digestive tracts were designed for slow, continuous feeding rather than the 2-3 meals per day the domestic lifestyle has normalized. While constant feeding isn't practical, the more you can spread out the number of feedings the better.

WILDWOOD FARM AND TRIPLE CROWN FEEDS.

Our partnership with Triple Crown began in 2014 through a promotion with the USEF encouraging farm members to compare their current feeding programs with Triple Crown products. We have found the TC products to be superior to other products primarily because of the EquiMix technology and the research support of a leading-edge team including independent representatives of Equine Universities, Medical clinics and top-level riders and trainers

Meet CJ SUPER FLY

The little mare that we now call Contessa, came to Wildwood Farm in July of 2025 with the name of Taco – definitely not a name that fit her.

Contessa is a registered American Shetland Pony, which differs from the traditional Shetland pony as the American has been infused with hackney characteristics such as highly animated gaits and upright neck and shoulders. These little ponies are bred for the showring, specifically in hand and harness.

We found Contessa at a local auction house and were taken by her statuesque beauty and truly miniature details. She was the epitome of a miniature HORSE, not pony. When she walked into the auction paddock, she had a high-stepping gait and looked very kind, even though she was pretty scared. The auctioneer said she had been started for 45 days, and the owners were selling her because she was too bossy to the other horses AND they had purchased her for their 6-year-old son, and he had lost interest. We were sure she would go for a lot of money, all the horses that day were well into the \$2000- \$3000 mark, and they were

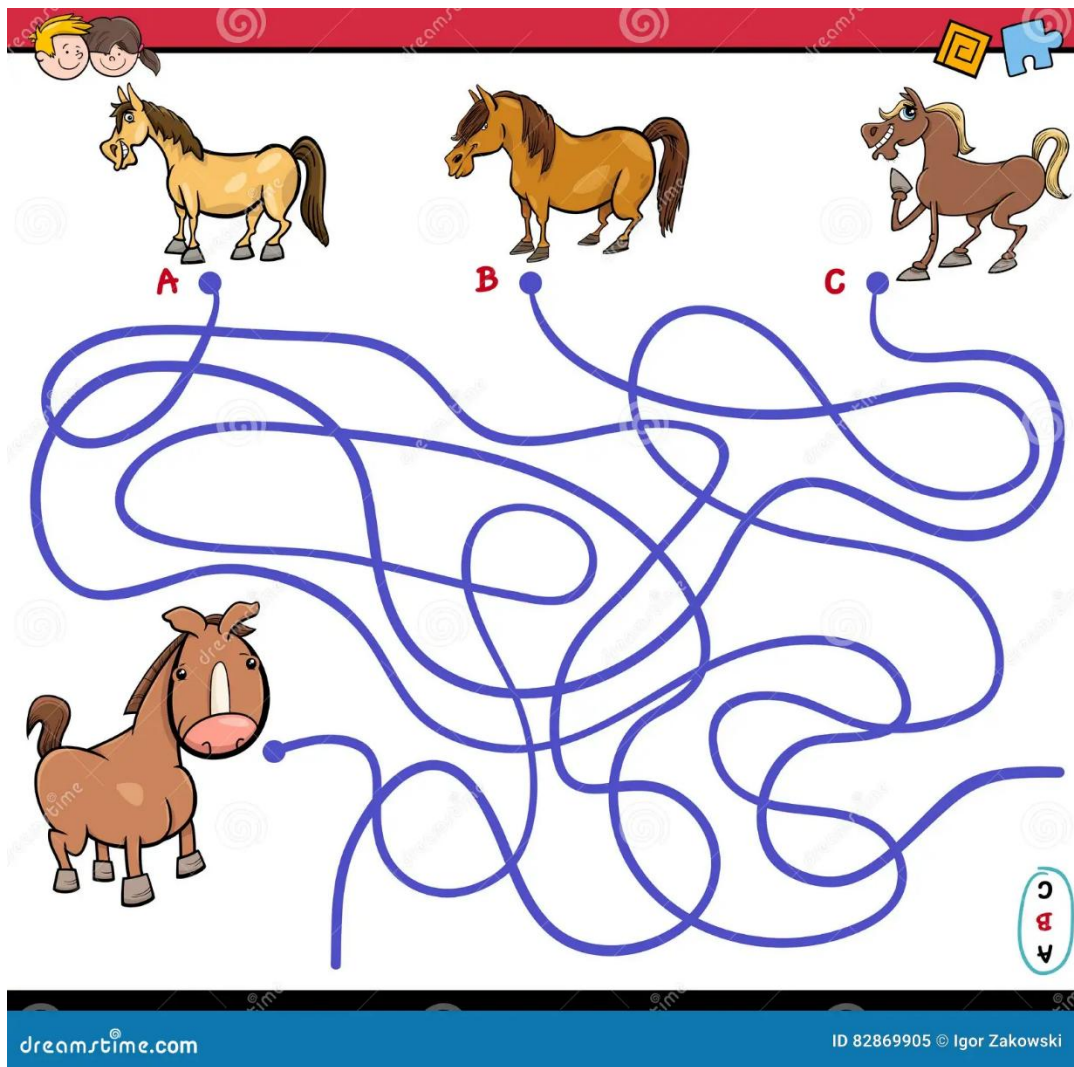


Not nearly as stunning as she was.

When the bidding was fading out at \$600, we had to throw our bid in and, as it turned out, we soon owned a new pony. We could not pick her up until the following day and though we hated the thought that she had to stay at the auction house overnight, we were just too far away to get her that night.

The next day she loaded beautifully and was soon on her way to Wildwood Farm. When she arrived, she met our mini ponies who were to be her barn mates, and settled in. It did not take her long to get on board with the routine and start feeling safe.

We don't know this little girl's history or why we are her 5th owners, but we are starting from scratch and looking forward to watching her blossom into a happy, confident little show pony!



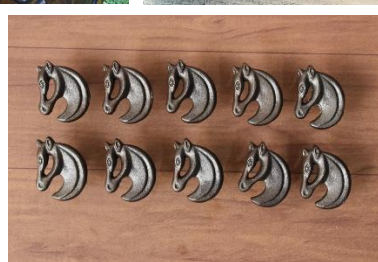
HAPPY
BIRTHDAY

We celebrate these Birthdays in September!

PEOPLE

Jesus September 4th, Karen Mulhern September 24th

FABULOUS EQUESTRIAN DRAWER PULLS!



Faroe Island

The Nykur of Sørvágsvatn

In Faroese legend, this sinister horse-shaped beast lures unsuspecting passersby only to drown them in the lake.



A Nykur is a mythical beast of Scandinavian folklore. It's often depicted as an aquatic monster that takes the shape of a gray, horse-like creature with inverted hoofs. The Nykur generally appears on the lakeshore, with half its body in the water, and looks to be quite tame to its unsuspecting victims.

The legendary Nykur (also known as the "Nix" in English) uses its beauty to lure passersby to pet and mount it, and once an unsuspecting man, woman, or child does so, the beast's "sticky skin" traps them in a terrifying barrel roll as it drags them down to the bottom of the lake to drown. This legend is mostly invoked by parents of small children, to scare them away from the edge of dangerous bodies of water.

The Nykur's one weakness is that if someone calls its name aloud, the creature will lose all of its power and retreat to the depths from which it came. And the Faroese legends say that once, a small child happened to see a Nykur roaming through the shallow water of Lake Sørvágsvatn while he was out playing. In awe of the creature's beauty, the child approached the Nykur. The child wanted his brother, Niklas, to see the pretty horse as well, so he called out to him. Since the child was still small and unable to speak properly, he accidentally shouted the word "Nykur" instead of "Niklas." Thus, having spoken the creature's name, it lost all its power and returned to the deep cold waters of lake Sørvágsvatn to try its luck another day.

On the island of Vágar in the Faroe Islands, a silver statue of the mythical Nykur rises from the northern tip of Lake Sørvágsvatn. The statue was created by local artist Pól Skarðenni, and appeared in the waters of the lake in 2017. Nearby, a plaque tells the legend of this sinister beast.

The Pintabian Horse



A relatively new breed that only appeared in 1992, the Pintabian is a unique crossbreed that has quickly trotted into the hearts of many horse enthusiasts.

The Pintabian horses were created through a deliberate breeding program which involves crossbreeding Arabian purebreds with tobiano-patterned horses. For a horse to be recognized as a Pintabian, it must have a tobiano pinto pattern and possess more than 99% Arabian bloodline.

Achieving this takes **eight** generations of careful breeding. Initially, a purebred Arabian is crossed with a tobiano horse of another breed. If the resulting foal also inherits the tobiano pattern, it is bred back to a purebred Arabian with the aim to produce another tobiano offspring.

The Pintabian horses embody the same ideal structure and shape desired in Arabian horses. A broad forehead, dainty muzzle, small ears, and an elegant body add to their alluring appearance. In terms of size, this breed typically stands between 14 and 15 hands high and weighs approximately 900 to 1100 lbs.

Now let's turn our attention to their striking markings. A true Pintabian horse will have beautiful, and entirely unique, tobiano markings over their entire body. Their base coat colors can range from black, bay, and buckskin to chestnut, dun, grey, grullo, and palomino.

Underneath their beautiful exterior is a spirited temperament that matches their fiery speed and agility. Just like their Arabian ancestors, they're hot-blooded and share the same athletic prowess and intelligence.

At its core, the Pintabian is a fascinating "designer breed." While purebred Arabians typically do not possess any pinto genes, the Pintabians inherit the beautiful pinto variations.

Cont'd from page 1

the researchers dig deeper.

Reading Through the Stop Sign

In horses and their relatives, this genetic stop sign isn't actually stopping anything. Instead, the cellular machinery ignores it and keeps reading, inserting a different building block (amino acid) than was originally there.

The modified KEAP1 becomes more responsive to stresses from burning oxygen, leading to increased protective activity. This creates more antioxidants that shield cells from damage while also boosting energy production.

The team confirmed these effects by comparing horse and mouse muscle cells. Horse cells burned significantly more oxygen and produced more energy. When exposed to harmful compounds, horse cells also proved much more resistant to damage than cells from other species.

To test whether this specific mutation directly caused these effects, the researchers created cell models comparing the original form of KEAP1 to the horse version. The horse version consistently activated more protective mechanisms and enhanced energy production.

What makes this genetic adaptation work? The researchers found that horses have additional mutations in the cellular machinery that reads genetic material. These mutations enhance the ability to read through stop signs, particularly at the KEAP1 gene.

This evolutionary solution gives horses a distinct edge: they can burn lots of oxygen for energy while simultaneously protecting themselves from the damage that would typically result. It's kind of like having a sports car with a built-in cooling system that prevents overheating.

The timing of this genetic innovation matches what we know about horse evolution. All modern horses, zebras, and donkeys share this mutation, suggesting it evolved in their common ancestor roughly 4-4.5 million years ago. This coincides with horses adapting to open grasslands and evolving single-toed feet, changes that enabled greater speed and stamina.

This type of genetic innovation was previously thought to happen mainly in viruses. This discovery shows that mammals can use similar strategies to evolve new traits.

Beyond explaining horse biology, this research might help medical science. The insights could guide treatments for conditions like respiratory diseases. Additionally, understanding how cells can read through genetic stop signs could help develop treatments for genetic diseases caused by similar mutations, which account for about 11% of human genetic disorders.

Through several coordinated molecular changes, horses developed a system that allows tremendous oxygen consumption while protecting their tissues from the resulting stress. This adaptation helped turn modest leaf-eaters into animals capable of sustained speed and endurance thanks to a genetic mishap that, ironically, accelerates their performance.

Methodology

The researchers compared KEAP1 genes from 196 mammal species to identify the unique stop codon in horses. They used RNA interference to silence KEAP1 in horse cells and observed increased NRF2 activity. Mass spectrometry confirmed the stop codon was being recoded to cysteine, creating a full-length protein. They measured oxygen consumption and ATP production in horse and mouse cells and created cellular models with either the ancestral or horse version of KEAP1 to directly test its effects.

Results

The scientists discovered that horses have a premature stop codon in the KEAP1 gene that gets recoded to cysteine

during protein production. This R15C mutation makes KEAP1 more sensitive to oxidative stress, leading to increased NRF2 activity. Horse cells showed significantly higher oxygen consumption, ATP production, and resistance to oxidative damage compared to other species. Horses also have specific mutations in SBP2 and eEFSEC proteins that enhance stop codon recoding.

Limitations

Since all other branches of the horse family are extinct, the researchers couldn't determine exactly when this mutation first appeared. The study primarily used cell cultures rather than measurements in live horses during exercise. While the research demonstrates benefits of the R15C mutation, it can't rule out potential downsides of persistently elevated NRF2 activity in certain contexts.

Discussion and Takeaways

This discovery reveals a novel mechanism of vertebrate adaptation through stop codon recoding – previously thought to occur mainly in viruses. The mutation provides horses with dual benefits: enhanced energy production and improved protection against oxidative damage. These findings may inform treatments for conditions involving oxidative stress and genetic diseases caused by premature stop codons, which affect approximately 11% of human genetic disorders.

Publication Information

The study, “Running a genetic stop sign accelerates oxygen metabolism and energy production in horses,” was authored by Gianni M. Castiglione and colleagues. It appeared in the March 28, 2025 issue of *Science* (Volume 387).

