Nitro PRO

A Neurotransmitter Formula using the Nitric Oxide Pathway

Introducing Nitro PRO created from Nobel Prize Winning Science

- Nitric Oxide promotes vasodilatation
- Nitric Oxide can increase blood flow & capillary perfusion to the equine hoof & laminae
- Nitric Oxide normalizes blood pressure, blood flow and glucose balance
- Nitric Oxide reduces stress and promotes a calming effect
- Nitric Oxide decreases inflammation and pain (eNOS)
- Nitric Oxide decreases healing time (the arginine solution) [1]

While controversy exist about the causation of laminitis, there is little question as to the role of compromised laminar bloodflow in the pathophysiology and management of acute and chronic laminitis. (LSU press release)[2] This has lead to efforts to improve laminar bloodflow via NO therapies. Nitro PRO is a bio identical NO therapy engineered to deliver clinically proven restorative NO precursor amino acids, neutralizing network antioxidants, regulating flavonoid, vitamins, minerals, and whole herbs to the laminae and hoof via endothelial Nitric Oxide Synthase (eNOS) mediated bloodflow (1998 Nobel Prize).

Nitric Oxide (eNOS) proven by the 1998 Nobel Prize winning team, is the definitive mechanism behind system wide vasodilatation, which promotes laminar blood flow, decreases laminar swelling, and supports recovery from laminitis. Concerns have arisen regarding drugs such as Nitroglycerin and sildenafil which are being clinically studied and used in the Equine arena for laminitis. These concerns regard unopposed free radical production, oxidative stress/tolerance (Nitroglycerin[3]), cGMP breakdown inhibition (sildenafil), and blood pooling at blood vessel lesion sites[4]. This has opened up the opportunity for a multi-nutrient Nutraceutical application engineered to produce Nitric Oxide, nature's nutrient delivery pathway, without the side effects related to the more popular pharmaceutical applications.

Nitro PRO is a product which strikes at the heart of laminitis pathophysiology.

Nitro PRO contains a proprietary blend of clinically established Nitric Oxide amino acid precursors[5] to include Arginine & Citrulline, network antioxidants[6] to include Vitamin C, Vitamin E, N-Acetyl-L Cysteine and flavonoid regulators[7] including Citrus Bio Flavonoid and Horse Chestnut seed extract.

Suggested Dosage: 1 scoop (32 cc per 1000lbs)
Top dress on feed 2 times a day for 10 days, then dose once a day until issue has been resolved.
Powder can be mixed with water in an oral syringe, dose into mouth slowly so horse can lap it up.
This prevents loss and insures horse gets full dose.

See dosage sheet for additional protocol options for severe laminitis.

Research Abstracts

Glyceryl trinitrate enhances nitric oxide mediated perfusion within the equine hoof.
J Endocrinol. 1996 Nov;151(2):R1-8. Hinckley KA, Fearn S, Howard BR, Henderson, IW.

Laminitis, a microvascular disease of the equine hoof leads to severe lameness. Exogenous iv 1-arginine and transdermal nitric oxide donors, such as GTN, applied to the pasterns improve lameness during acute laminitis. Near Infrared spectroscopy in an earlier study showed haemostasis and ischaemia in the hoof during acute laminitis, both were alleviated by 1-arginine. Quantitative NIRS in the present study shows that transdermal GTN increases blood flow in the equine hoof. It is concluded that glyceryl trinitrate enhances nitric oxide mediated perfusion within the equine hoof in normal and chronically laminitic horses and ponies.

Nitric oxide donors as treatment for grass induced acute laminitis in ponies.
Equine Vet J. 1996 Jan;28(1):17-28. Hinckley KA, Fearn S, Howard BR, Henderson IW.

The potential for participation of the arginine-nitric oxide system in the aetiology of acute equine laminitis has been assessed. Nitric oxide (NO), produced by the action of NO synthase (NOS) on its substrate l-arginine, relaxes vascular smooth muscle to cause vasodilation. An attenuated normal vasodilatory tone may characterise the pathogenesis of acute equine laminitis. An intravenous infusion of 10% l-arginine in 0.9% saline caused vasodilatation in the hoof of a normal pony and immediate reperfusion of laminal tissues in an acutely laminitic pony, detected noninvasively by near infrared spectroscopy (NIRS), but the amino acid had little effect on systemic blood pressure. Treatment of acute laminitis with glyceryl trinitrate applied topically to the pasterns reduced the typical 'bounding pulses' in treated limbs, reduced lameness and lowered systemic blood pressure. Nitric oxide is likely to participate in the multifactorial pathogenesis of equine laminitis.

Flavonoids Attenuate Cardiovascular Disease, Inhibit Phosphodiesterase, & Modulate Lipid Homeostasis in Adipose Tissue & Liver
Experimental Biology and Medicine 231:1287-1299 (2006). Peluso, M.

Plant flavonoids are widely distributed polyphenolic compounds of the human diet. They consist of six major classes based on specific structural differences: flavonols, flavones, flavanones, catechins, anthocyanidins, and isoflavones. All of the major classes of flavonoids are comprised of three six-membered rings: an aromatic A-ring fused to a heterocyclic C-ring that is attached through a single carbon-carbon bond to an aromatic Bring. Population studies have shown that flavonoid intake is inversely correlated with mortality from cardiovascular disease, and numerous flavonoids of dietary significance have been shown to beneficially impact parameters associated with atherosclerosis, including lipoprotein oxidation, blood platelet aggregation, and vascular reactivity. Therapeutic effects of flavonoids on platelet aggregability and blood pressure have been attributed to competitive inhibition of cyclic nucleotide phosphodiesterase (PDE), an elevation in cAMP level, and subsequent activation of protein kinase A (cAMP-dependent protein kinase). In addition, flavonoids may induce neutral lipid hydrolysis from lipid stores through PDE inhibition in adipose tissue and liver. Indeed, the three-dimensional structure of many flavonoids is sterically and electrostatically compatible with the catalytic site of cAMP PDE3 and PDE4. Flavonoids have also been reported to suppress pathways of lipid biosynthesis and of very low-density lipoprotein production in cultured hepatocytes. Continued studies of the biochemical mechanisms underlying the biological effects of plant flavonoids may uncover new strategies for the treatment of cardiovascular disease, as well as associated conditions such as obesity, hepatic steatosis, and Type 2 diabetes.

References:

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2. University Of Illinois At Chicago (2003, January 10). Viagra Deaths Explained By New Understanding Of Platelet Clumping. *ScienceDaily*. Retrieved June 8, 2009, from http://www.sciencedaily.com /releases/2003/01/030110193129.htm
3. Hirai, N., et al. 2003. Attenuation of Nitrite Tolerance and Oxidative Stress by an Angiotensin II Receptor Blocker in Patients with Coronary Spastic Angina. *Circulation* 2003; 108: 1445-1450.
4. Chambers, J.C., et al. 1999. Demonstration of rapid onset vascular endothelial dysfunction after hyperhomocysteinemia: An effect reversible with Vitamin C therapy. *Circulation* 99*:* 1156-60.
5. Taddei, S. et al. 1998. Vitamin C improves endothelium-dependent vasodilation by restoring nitric oxide activity in essential hypertension. *Circulation* 97: 2222-29.
6. Pittler, M.H. , and E. Ernst. 1998. Horse Chestnut Seed extract for chronic venous insufficiency – a criteria-based systemic review. *Arch Dermatol* 134:1356-60.