How to best support equine immune system function is a very common concern. There are two mistakes/misconceptions many people have. One is that you should try to “stimulate”. The other is that stimulating is always a good thing.

Trying to stimulate the immune system is futile if it does not have the basic raw materials it needs to function – nutrients. Hydration, calories, protein/amino acids, fatty acids, vitamin and mineral status all influence immune function. These are the fuels and also regulate and orchestrate right down to the level of DNA.

For example, this recent study in humans found clear effects on immune function with even mildly inadequate zinc intakes that did not produce any change in zinc blood levels:

Proc Natl Acad Sci U S A. 2011 Dec 27;108(52):20970-5. doi: 10.1073/pnas.1117207108. Epub 2011 Dec 14.

Genomic analysis, cytokine expression, and microRNA profiling reveal biomarkers of human dietary zinc depletion and homeostasis.

Ryu MS[1], Langkamp-Henken B, Chang SM, Shankar MN, Cousins RJ.

Author information

Abstract

Implementation of zinc interventions for subjects suspected of being zinc-deficient is a global need, but is limited due to the absence of reliable biomarkers. To discover molecular signatures of human zinc deficiency, a combination of transcriptome, cytokine, and microRNA analyses was applied to a dietary zinc depletion/repletion protocol with young male human subjects. Concomitant with a decrease in serum zinc concentration, changes in buccal and blood gene transcripts related to zinc homeostasis occurred with zinc depletion. Microarray analyses of whole blood RNA revealed zinc-responsive genes, particularly, those associated with cell cycle regulation and immunity. Responses of potential signature genes of dietary zinc depletion were further assessed by quantitative real-time PCR. The diagnostic properties of specific serum microRNAs for dietary zinc deficiency were identified by acute responses to zinc depletion, which were reversible by subsequent zinc repletion. Depression of immune-stimulated TNFα secretion by blood cells was observed after low zinc consumption and may serve as a functional biomarker. Our findings introduce numerous novel candidate biomarkers for dietary zinc status assessment using a variety of contemporary technologies and which identify changes that occur prior to or with greater sensitivity than the serum zinc concentration which represents the current zinc status assessment marker. In addition, the results of gene network analysis reveal potential clinical outcomes attributable to suboptimal zinc intake including immune function defects and predisposition to cancer. These demonstrate through a controlled depletion/repletion dietary protocol that the illusive zinc biomarker(s) can be identified and applied to assessment and intervention strategies.

I'll give more details regarding basic nutrients below, but want to emphasize a very important point here. There is *nothing* that will substitute for inadequate levels of a basic nutrient. Their jobs in signaling between cells, forming antibodies and cytokines, and protecting the immune system cannot be bypassed by any herb, isolated other nutrients, drug or vaccines. Nutrition must come first.

Major Components of the Immune System

Th2: This is the “primitive” immune system. It does not require antibodies or lymphocytes with “memory” or previous invasions to work. Foreign organisms/substances, damaged or abnormal body cells, parasites will all trigger a Th2 response which is primarily inflammatory. Allergies are also predominantly Th2 responses. A integral component is the complement system (below).

Th1: Th1 is the adaptive immune response. It involves for formation of various classes of antibodies specific to a particular organism. Memory T cells are also formed, which are cells which will remember this organism and can produce antibodies much faster than possible on the first exposure.

Complement system: A family of circulating proteins which are part of the Th2 immune response. They attract white blood cells to foreign organism and enhance them being destroyed by white blood cells. Malfunctioning in this system may also be involved in autoimmune diseases.+ Cytokines: Cytokines are small proteins secreted by nervous system and immune system cells which trigger reactions in other cells types. Some are inflammatory, others counter-regulatory/anti-inflammatory. Antibodies: Antibodies are proteins produced by a specific class of white blood cells. They bind to organisms, or cells identified as infected by organisms, targeting them for destruction.

Protein and Immune Function

Protein plays a critical role in immune function. Antibodies, cytokines and the complement system are all manufactured from protein. For example:

J Immunol. 2012 Jan 1;188(1):77-84. doi: 10.4049/jimmunol.1004027. Epub 2011 Nov 23.

Protein energy malnutrition impairs homeostatic proliferation of memory CD8 T cells.

Iyer SS[1], Chatraw JH, Tan WG, Wherry EJ, Becker TC, Ahmed R, Kapasi ZF.

Author information

Abstract

Nutrition is a critical but poorly understood determinant of immunity. There is abundant epidemiological evidence linking protein malnutrition to impaired vaccine efficacy and increased susceptibility to infections; yet, the role of dietary protein in immune memory homeostasis remains poorly understood. In this study, we show that protein-energy malnutrition induced in mice by low-protein (LP) feeding has a detrimental impact on CD8 memory. Relative to adequate protein (AP)-fed controls, LP feeding in lymphocytic choriomeningitis virus (LCMV)-immune mice resulted in a 2-fold decrease in LCMV-specific CD8 memory T cells. Adoptive transfer of memory cells, labeled with a division tracking dye, from AP mice into naive LP or AP mice demonstrated that protein-energy malnutrition caused profound defects in homeostatic proliferation. Remarkably, this defect occurred despite the lymphopenic environment in LP hosts. Whereas Ag-specific memory cells in LP and AP hosts were phenotypically similar, memory cells in LP hosts were markedly less responsive to polyinosinic-polycytidylic acid-induced acute proliferative signals. Furthermore, upon recall, memory cells in LP hosts displayed reduced proliferation and protection from challenge with LCMV-clone 13, resulting in impaired viral clearance in the liver. The findings show a metabolic requirement of dietary protein in sustaining functional CD8 memory and suggest that interventions to optimize dietary protein intake may improve vaccine efficacy in malnourished individuals.

In addition to protein in general, specific amino acids (the building blocks of protein) play critical roles Glutamine, an amino acid, is both a fuel for immune system cells and a precursor for glutathione, which is a critical intracellular antioxidant that protects immune system cells from damage from “friendly fire” when they are destroying organisms.

> Amino Acids. 1999;17(3):227-41. Glutamine and the immune system. Calder PC,Yaqoob P. Institute of Human Nutrition, University of Southampton, United Kingdom Glutamine is utilised at a high rate by cells of the immune system in culture and is required to support optimal lymphocyte proliferation and production of cytokines by lymphocytes and macrophages. Macrophage-mediated phagocytosis is influenced by glutamine availability. Hydrolysable glutamine dipeptides can substitute for glutamine to support in vitro lymphocyte and macrophage functions. In man plasma and skeletal muscle glutamine levels are lowered by sepsis, injury, burns, surgery and endurance exercise and in the overtrained athlete. The lowered plasma glutamine concentrations are most likely the result of demand for glutaminne (by the liver, kidney, gut and immune system) exceeding the supply (from the diet and from muscle). It has been suggested that the lowered plasma glutamine concentration contributes, at least in part, to the immunosuppression which

accompanies such situations. Animal studies have shown that precursors has been provided, usually by the parenteral route, to patients following surgery, radiation treatment or bone marrow transplantation or suffering from injury. In most cases the intention was not to stimulate the immune system but rather to maintain nitrogen balance, muscle mass and/or gut integrity. Nevertheless, the maintenance of plasma glutamine concentrations in such a group of patients very much at risk of immunosuppression has the added benefit of maintaining immune function. Indeed, the provision of glutamine to patients following bone marrow transplantation resulted in a lower level of infection and a shorter stay in hospital than for patients receiving glutamine-free parenteral nutrition. inclusion of glutamine in the diet increases survival to a bacterial challenge.

Supporting the Ill or Injured Horse

For support of the ill or injured horse, 15 to 30 mg/kg of L-Glutamine on top of the maintenance requirement is reasonable.

There's a time and a place for arginine supplementation as well, but it isn't always easy to know when. Arginine is contraindicated in horses that are septic from bacterial infections, i.e. have fevers related to a bacterial infection. Arginine can also "feed" some viruses,

(e.g. Herpes) and make the symptoms of both viral and bacterial infections far worse in the initial stages. However, because of arginine's key role in fueling basic immune system functions, you do want to make sure to provide at least what the horse's baseline intake would be.

The arginine level in grass hays averages 4.5% of the crude protein. Grains and by-products contain a maximum of 1% of the crude protein. Using these figures, you can determine approximately how much arginine your horse would be taking in at maintenance and how far short the horse is if he is not eating a full ration.

For example, a horse eating 10 kg of a 10% protein hay is taking in 1000 grams of protein, 45 grams of which will be arginine. If the horse is only eating half that much because he is ill or injured, you can supplement with 22.5 grams of arginine divided over the day. Always provide arginine in divided doses and with a meal or syringed in with protein. This will avoid a "rush" of nitric oxide production.

Once fever has broken or the horse is past the first 3 to 7 days of an injury, an additional 10 grams of arginine per 500 lbs of bodyweight/day, divided between meals, is reasonable to support healing.

Dehydration has a negative effect on immune function when the body is subjected to stress, such as by exercise:

Int J Sports Physiol Perform. 2008 Dec;3(4):531-46.

Effect of hydration status on high-intensity rowing performance and immune function.

Penkman MA[1], Field CJ, Sellar CM, Harber VJ, Bell GJ.

Author information

Abstract

PURPOSE: This study determined the effect of dehydration and rehydration (DR) on performance, immune cell response, and tympanic temperature after high-intensity rowing exercise.

METHODS: Seven oarswomen completed two simulated 2000-m rowing race trials separated by 72 h in a random, cross-over design. One trial was completed in a euhydrated (E) condition and the other using a DR protocol.

RESULTS: The DR condition resulted in a 3.33+/-0.14% reduction in body mass (P<.05) over a 24-h period followed by a 2-h rehydration period immediately before the simulated rowing race. There was a greater change in tympanic temperature observed in the DR trial (P<.05). There were increases in the blood concentration of leukocytes, lymphocytes, lymphocyte subsets (CD3+, CD3+/4+, CD3+/8+, CD3-/16+, CD4+/25+; P<.05) and decreases in lymphocyte proliferation and neutrophil oxidative burst activity immediately following the simulated race (P<.05) in both trials. Blood leukocyte and neutrophil concentrations were greater after exercise in the DR trial (P<.05). Whereas most immune measures returned to resting values after 60 min of recovery in both trials, lymphocyte proliferation and the concentrations of CD3+/4+ and CD4+/25+ cells were significantly lower than before exercise. Blood leukocyte and neutrophil concentrations were significantly higher before and after exercise in the E trial.

CONCLUSION: The effects of dehydration/rehydration did not negatively influence simulated 2000-m rowing race performance in lightweight oarswomen but did produce a higher tympanic temperature and had a differential effect on blood leukocyte, neutrophil, and natural killer (CD3-/16+) cell concentrations after exercise compared with the euhydrated state.

Minerals and their effect on the Horse Immune System

Getting back to minerals, as with zinc, virtually all minerals have documented effects on the immune system. In this study, magnesium and manganese had anti-inflammatory effects while enhancing other aspects of the immune response:

Arch Pharm Res. 2007 Jun;30(6):743-9.

Effects of supplementation with higher levels of manganese and magnesium on immune function.

Son EW[1], Lee SR, Choi HS, Koo HJ, Huh JE, Kim MH, Pyo S.

Author information

Abstract

The magnesium (Mg) and manganese (Mn) were evaluated for its effectiveness as an immunomodulator in rats. The treatments were as follows: Group 1, AIN-93M diet (0.05% Mg, 0.001% Mn); Group 2, high-dose Mg (0.1% Mg, 0.001% Mn); and Group 3, high dose Mn (0.05% Mg, 0.01% Mn) (n-12/group). After 12 weeks of supplementation, rats were sacrificed to assess the effect on a range of innate responses (tumoricidal activity, oxidative burst and nitric oxide) and the mitogen-stimulated lymphoproliferative response. Immune function was significantly affected in both the high dose Mg and the Mn group. Lymphocyte proliferative responses and NK cell activity were measured in pooled spleen from each group. The mitogen response of lymphocytes to LPS in the spleen was significantly reduced in high dose Mg-treated groups, whereas the response to ConA was not affected in both high dose minerals-treated groups. The reactive oxygen species level of macrophages was decreased in both groups. These effects were more pronounced in high dose Mg-treated group. Nitric oxide production was also decreased in high dose minerals-treated group. In addition, tumoricidal activities of splenic NK cell and peritoneal macrophage in mineral exposed rats were significantly increased. Moreover, percent death of macrophage was reduced in two groups receiving high dose mineral supplements. Taken together, the present data suggest that high dose trace min erals exert a differential effect on the function of immune cells.

There are almost 100 formal studies on the effects of magnesium status alone on inflammation and allergy. Both copper and zinc are essential cofactors in the important antioxidant enzyme system superoxide dismutase. As with zinc above, copper also has a

direct effect on immune function:

Gen Physiol Biophys. 2000 Mar;19(1):49-58.

Bovine monocyte-derived macrophage function in induced copper deficiency.

Cerone S[1], Sansinanea A, Streitenberger S, García C, Auza N.

Author information

Abstract

The effect of molybdenum-induced copper deficiency on monocyte-derived macrophage function was examined. Five female calves were given molybdenum (30 ppm) and sulphate (225 ppm) to induce experimental secondary copper deficiency. Oxidant production by bovine macrophages was measured after stimulation with phorbol myristate acetate (PMA) and opsonized zymosan (OpZ). Lipoperoxidative effects inside of macrophage, superoxide dismutase activity, superoxide anion and hydrogen peroxide formation were determined. Copper deficiency was confirmed from decreased serum copper levels, and animals with values less than 5.9 micromol/l were considered deficient. The content of intracellular copper decreased about 40% in deficient cells compared with the controls. The respiratory burst activity determined by nitroblue tetrazolium reduction was significantly impaired with both stimulants used. Superoxide anion formation was less affected than hydrogen peroxide generation. In addition, increased lipid peroxidation was observed. It could be concluded that the effect of these changes may impair the monocyte-derived macrophage function in the immune system.

J Nutr. 2000 Jun;130(6):1536-42.

Copper deficiency suppresses effector activities of differentiated U937 cells.

Huang ZL[1], Failla ML.

Author information

Abstract

Dietary copper (Cu) deficiency impairs both innate and acquired branches of immunity. Specific roles of Cu in the activation and effector activities of host-defense cells remain largely unknown. The effects of Cu status on effector activities of a monocytic cell line were investigated as an initial step in the elucidation of specific functions of Cu in phagocytic cells. Exposure of differentiating U937 human promonocytic cells to 5 micromol/L 2,3, 2-tetraamine (tet), a high affinity Cu chelator, for 4 d decreased cellular Cu by 62% without altering cellular Cu,Zn-superoxide dismutase (SOD) activity, Zn content, mitochondrial activity and protein synthesis. In contrast, Cu deficiency suppressed the respiratory burst activity and markedly compromised the ability of U937 cells to kill Salmonella. Similarly, treatment of RAW264.7 murine macrophages with 5 micromol/L tet decreased cell Cu by 78% and Cu,Zn-SOD activity by 15% and increased bacterial survival by 180%. The tet-induced impairment of respiratory burst and bactericidal activities was blocked in cultures supplemented with Cu, but not Zn or Fe. In addition, lipopolysaccharide (LPS)-induced secretion of the inflammatory mediators, tumor necrosis factor-alpha, interleukin (IL)-1beta, IL-6 and prostaglandin E(2) (PGE(2)), was decreased by 30-60% in tet-treated U937 cells. Flow cytometric analysis of the surface antigens CD11b and CD71 showed that the suppressed activities of Cu-deficient cells were not due to an attenuation in the degree of differentiation or secondary iron deficiency. These data demonstrate that U937 cells provide a useful model for examining the biochemical roles of Cu in monocyte activity.

Essential Fatty Acids in a Healthy Immune System

Essential fatty acids are also key players. This is the omega-3 and omega-6 group of fatty acids. They are called “essential” because the horse’s body cannot manufacture them and they must be present in the diet. Although a bit of an oversimplification, the omega-6 fatty acids are instrumental in inflammatory/innate immune reactions while the omega-3s support the counter-regulatory and anti-inflammatory responses that keep those in check.

Inadequate omega-3 intake compared to omega-6 has been implicated in everything from problems in fetal brain development to heart disease in humans. It’s a human problem because of inadequate intakes of fresh vegetables/fruits and whole grains, as well as

meats and eggs from intensively raised animals that contain omega-6 to omega-3 ratios as high as 20:1 compared to the natural levels of about 4:1 in naturally fed animals.

The situation is similar in horses. Fresh grass/vegetation has an omega-6 to omega-3 ratio of about 1:4. However, when grass is cut and cured for hay, the omega-3s are lost. Grains, vegetable oils and popular seed meals like soybean or sunflower also have high ratios of omega-6 to omega-3 fats.

While horses and people have different natural intakes from their natural diets (horses 1:4, humans 4:1), in both cases there is an overabundance of omega-6. Several equine studies have shown how omega-3 fatty acids can modify inflammatory reactions:

Am J Vet Res. 2005 Sep;66(9):1503-8.

Effects of the omega-3 fatty acid, alpha-linolenic acid, on lipopolysaccharide-challenged synovial explants from horses.

Munsterman AS[1], Bertone AL, Zachos TA, Weisbrode SE.

Author information

Abstract

OBJECTIVE: To determine the effects of pretreatment with alpha-linolenic acid, an omega-3 polyunsaturated fatty acid, on equine synovial explants challenged with lipopolysaccharide (LPS).

ANIMALS: 8 mature mixed-breed horses (4 mares and 4 geldings).

PROCEDURE: Synovial explants were assigned to receive 1 of 7 concentrations of alpha-linolenic acid, ranging from 0 to 300 microg/mL. At each concentration, half of the explants were controls and half were challenged with 0.003 microg of LPS as a model of synovial inflammation. Cell inflammatory response was evaluated by measurement of prostaglandin E2 production via an ELISA. Synovial cell viability, function, histomorphologic characteristics, and cell membrane composition were evaluated by use of trypan blue dye exclusion, hexuronic acid assay for hyaluronic acid, objective microscopic scoring, and high-performance liquid chromatography, respectively.

RESULTS: Challenge with LPS significantly increased production of prostaglandin E2 and decreased production of hyaluronic acid. Treatment with alpha-linolenic acid at the highest dose inhibited prostaglandin E2 production. Cell viability and histomorphologic characteristics were not altered by treatment with alpha-linolenic acid or LPS challenge. Treatment with alpha-linolenic acid increased the percentage of this fatty acid in the explant cell membranes.

CONCLUSIONS AND CLINICAL RELEVANCE: Results suggest that investigation of alpha-linolenic acid as an anti-inflammatory medication for equine synovitis is warranted.

Shock. 2000 Aug;14(2):222-8.

Effect of intravenous infusion of omega-3 and omega-6 lipid emulsions on equine monocyte fatty acid composition and inflammatory mediator production in vitro.

McCann ME[1], Moore JN, Carrick JB, Barton MH.

Author information

Abstract

The effect of intravenous administration of lipid emulsions enriched with omega-3 (n3) and omega-6 (n6) fatty acids on equine monocyte phospholipid fatty acid composition and the synthesis of inflammatory mediators in vitro was evaluated. In a randomized crossover design, horses were infused intravenously with 20% lipid emulsions containing n3 or n6 fatty acids. Monocytes were isolated from the horses before and 0 h, 8 h, 24 h, and 7 days after lipid infusion. Monocyte fatty acid analysis demonstrated incorporation of the parenteral n3 and n6 fatty acids in monocyte phospholipids immediately after infusion, with changes in the fatty acid composition persisting for up to 7 days after infusion. In vitro production of the inflammatory mediators thromboxane B2/thromboxane B3 (TXB(2/3)) and tumor necrosis factor-alpha (TNFalpha) by peripheral blood monocytes was diminished by n3 lipid infusion and was unchanged or increased by n6 lipid infusion. The results of this study demonstrate that short-term infusions of n3 and n6 fatty acid-enriched lipid emulsions alter the fatty acid composition of equine monocyte phospholipids and modify the inflammatory response of these cells in vitro. These results also support further investigation into the use of parenteral n3 fatty acids as part of the supportive therapy of patients with multiple organ dysfunction (MODS) or systemic inflammatory response syndrome (SIRS).

Selenium and Vitamin E for a Healthy Immune System

Selenium and vitamin E are best known for protecting cells from oxidative stress induced by exercise, immune system destruction of organisms, or environmental toxins. However, again there is also another direct role in supporting the activity of the immune system, as in these studies on selenium and vitamin E:

Indian J Exp Biol. 1998 Feb;36(2):203-5.

Effect of selenium deficiency and its supplementation on DTH response, antibody forming cells and antibody titre.

Kukreja R[1], Khan A.

Author information

Abstract

Selenium levels and the activity of Selenoenzyme glutathione peroxidase were measure in whole blood in order to assess the selenium status. Delayed type of hypersensitivity (DTH) reaction was suppressed significantly in selenium deficient rats indicating the decrease in cellular immunity. The B cell function was impaired in selenium deficient rats as evident from decrease in the number of plaque forming cells and antibody titre. Selenium supplementation for 30 days recovered the DTH response and B cell function to a marked extent.

Vitam Horm. 2011;86:179-215. doi: 10.1016/B978-0-12-386960-9.00008-3.

Vitamin E and immunity.

Pekmezci D[1].

Author information

Abstract

Vitamin E is the most important chain-breaking, lipid-soluble antioxidant present in body tissues of all cells and is considered the first line of defense against lipid peroxidation and it is important for normal function of the immune cells. However, vitamin E deficiency is rare in well-nourished healthy subjects and is not a problem, even among people living on relatively poor diets, both T- and B-cell functions are impaired by vitamin E deficiency. While immune cells are particularly enriched in vitamin E because of their high polyunsaturated fatty acid content, this point puts them at especially high risk for oxidative damage. Besides its immunomodulatory effects, vitamin E also plays an important role in carcinogenesis with its antioxidant properties against cancer, and ischemic heart disease with limiting the progression of atherosclerosis. Supplementation of vitamin E significantly enhances both cell mediated and humoral immune functions in humans, especially in the elderly and animals.

Vitam Horm. 2000;59:305-36.

Vitamin E and immunity.

Moriguchi S[1], Muraga M.

Author information

Abstract

Vitamin E is a potent antioxidant and has an ability to modulate host immune functions. This chapter consists of five parts: (1) vitamin E deficiency and immunity, (2) vitamin E supplementation and immunity, (3) vitamin E and the decreased cellular immunity with aging, (4) vitamin E and T-cell differentiation in the thymus, and (5) vitamin E and acquired immune deficiency syndrome (AIDS). In vitamin E deficiency most of the immune parameters show a downward trend, which is associated with increased infectious diseases and the incidence of tumors. In contrast, vitamin E supplementation has various beneficial effects on the host immune system. The decreased cellular immunity with aging or during the development of AIDS is markedly improved by the intake of a high vitamin E diet. In addition, vitamin E plays an important role in the differentiation of immature T cells in thymus. Vitamin E deficiency induces the decreased differentiation of immature T cells, which results in the early decrease of cellular immunity with aging in spontaneously hypertensive rats. Conversely, vitamin E supplementation induces a higher differentiation of immature T cells via increased positive selection by thymic epithelial cells, which results in the improvement of decreased cellular immunity in the aged. Furthermore, vitamin E supplementation induces the early recovery of thymic atrophy following X-ray irradiation. Taken together, these results suggest that vitamin E is an important nutrient for maintaining the immune system, especially in the aged.

These are just a few of hundreds of studies linking nutrients to normal performance of the immune system. As researchers uncover more sensitive ways of testing, such as DNA expression, it becomes obvious that even relatively minor shortfalls of essential nutrients can impact the immune system.

Iron and the Immune System

Iron on the other hand can have very negative effects on inflammation, malignancy and in fighting infections. In fact, the body's natural response to these conditions is to rev up production of it's iron-trapping protein, ferritin, and lowering levels of circulating iron.

Iron supplementation has *no* place in the support of an ill or injured horse.

As above, there is no substitute for skipping the step of an adequate diet. Hay analysis to guarantee adequate protein and calorie intake, plus balancing of minerals, is an important first step. If you absolutely can't analyse, buy only very high quality, green, fragrant hay and consider feeding 5 to 10% alfalfa as well. For most of the UK and parts of Eurpoe *(information from Forageplus)*, high managanese and/or iron are likely to be problems, meaning you should avoid supplementing those.

Levels of Copper and Zinc to Feed Horses

Copper 300 to 400 mg/day and zinc 900 to 1200 mg/day, as sulphate or proteinate forms, are reasonable levels although, again, no guarantees that is correct without analysis. In many cases higher levels of copper and zinc are needed to balance the high levels of the antagonist minerals iron and manganese.

Selenium Supplementation for Horses

It is unusual to find adequate levels of selenium. You can best check for this with a whole blood selenium assay. In other areas, supplementation of 1/2 mg/day from selenium yeast is advisable depending upon horse work levels.

Supplementation of iodine at 2 to 2.5 mg/500 lbs bodyweight rounds out the picture.

Horses *not* on pasture need supplemental vitamin E (minimum 1000 IU/220 kgs in healthy horses) and micronised linseed for correct omega-6 and omega-3 ratios (100 – 200 grams per day).

Correct Nutrition for your Horse

Correct nutrition can reduce the risk or severity of infections, improve immune responses to vaccine and eventually modulate inflammatory reactions and allergies.

No horse will ever be completely immune to infections, let alone free from injuries, so the next issue to tackle is how to support the horse is he/she does become ill or injured.

The first step for doing that is to have a nutritionally adequate and properly balanced diet with 'forage focused' minerals at least 150% of NRC (National Research Council). Keeping the horse's body adequately nourished is extremely important to supporting the immune system. If the horse is already ill or injured and is off feed, do a calorie and protein count,

and be sure to scale back your balancing minerals to match intake.

If you are meeting mineral needs but still coming up short on protein, I would advise using a whey protein concentrate or isolate to make up the difference. Sixty grams of whey per day has generous branched chain amino acids (BCAAs) to support glutamine, and good levels of glutamine itself. Ot your can feed an Essential Amino Acid mix.

Equine Infection Fighting Support Summary

If the horse is not eating a normal amount, calculate calorie, protein and mineral intake
Make up at least the protein and minerals using a palatable balanced protein and mineral supplement.
Calculate arginine intake and supplement up to maintenance requirements. Feeding additional arginine (up to 10 grams daily), may be useful after fever (infections) or initial inflammation (wounds) has resolved.
Support glutamine levels with 10 to 20 grams of mixed BCAAs (450 kg horse) on top of maintenance requirements or 15 to 30 mg/kg of L-glutamine.

Herbs to Support the Horse Immune System

It would be impossible in the framework of this article to do a through discussion of herbs with effects on the immune system. But as a brief overview, with a few exceptions, the herbs used for immune system effects, such as Echinacea, work by themselves triggering an immune reaction, usually of the innate, Th2 immune system.

Although often used in hopes of helping the horse to fight an infection, it is important to remember that the infection itself is already “stimulating” the immune system. An enhanced Th2 response may help clear a extracellular bacterial infection, but not viral or intracellular bacteria (e.g. Lyme).

Adaptogens to Support the Horse Immune System

The adaptogens, in particular Ginseng, show the most promise in terms of capacity to stimulate both arms of the immune system, but much of the research is done with isolated ginsenosides rather than whole herb. We also need to remember that the ginsenoside profile of any given batch of herb will depend on where it was grown and the growing

conditions. This makes the use of herbs a bit of a crap shoot, especially with intracellular organisms where overactivation of the Th2 system may suppress the Th1, cell-mediated response needed to actually clear the organism.

All horses with immune system issues will benefit from balanced minerals being added to their diet. This 'forage focused' approach will support and maintain health in an intelligent way. To find out more about the benefits of feeding 'forage focused' minerals to your horse read this article.

help your horse maintain optimal health.

