Equine Dermatology - Selected Topics

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Introduction

The knowledge and experience in dealing with equine skin disease has expanded in the last decade and has become a very important subspecialty in veterinary dermatology. Updates on allergic hypersensitivities (Insect, adverse food reactions and atopic dermatitis) and common equine infectious diseases (pyoderma, dermatophyte and Malassezia) will be covered. A case orientated approach will be given with emphasis on clinical, diagnosis and therapeutic options.

Allergic hypersensitivities

The horse suffers from a variety of allergic hypersensitivity conditions. Insect hypersensitivity is the best defined and understood, particularly Culicoides hypersensitivity. Atopic dermatitis is also becoming a more commonly recognized entity in the horse. Food allergies are occasionally seen but are difficult to identify with certainty. Some conditions can present with either pruritus and/or hives and may create other generalized skin eruptions such as papules, scales and crusting.

Horses can mount an immediate hypersensitivity response and equine immunoglobulin IgE has been identified and characterized.[1-5] In horses, a single gene encoding the IgE heavy chain constant region (IGHE gene) exists per haploid genome and several allelic variants of the equine IGHE gene were found. IgE occurs in its soluble form in equine serum and physiological concentrations of total IgE are around 1000-fold higher in normal horse than in normal human serum. Maternal IgE is enriched in the colostrum and transferred to the neonatal foal after birth. Foals do not produce detectable concentrations of endogenous IgE for several months after birth. IgE-mediated mechanisms have been implicated in the pathogenesis of several allergic diseases in horses. The findings are mainly based on the induction of immediate skin reactions after intradermal testing with allergen extracts.[4]

Insect hypersensitivity: Insect hypersensitivity is the most common cause of equine pruritus. It is generally a seasonal highly pruritic dermatitis that can also involve urticaria. It is usually due to a hypersensitivity to salivary antigens administered by the biting insect. The most common insects involved include Culicoides, black flies, horn flies and stable flies. Occasionally we can also see reactions from mosquitoes, deer flies and horse flies. Like canine allergic dermatitis, both inhalation and percutaneous absorption of insect allergens most likely exist. Black ants, housefly, caddisfly and mayfly, dust and storage mites are insects that non-biting that may create this type of hypersensitivity. Clinical evidence strongly suggests that this disorder has a familial and therefore genetic tendency exists but other factors such as environment and lack of exposure at an early age are also important predisposing factors. Insect hypersensitivity has been shown to have IgE-induced immediate and late-phase reaction as well as cell-mediated delayed reactions. A complex interaction of inflammatory cells and their mediators produce this reaction. Eosinophils and lymphocytes constitute the major

inflammatory cells with increased numbers of CD5+ and CD4+ T lymphocytes and Langerhans' cells as well as LTB4 and LTD4 in affected skin lesions. IgE, IgE mRNA positive cells and tryptase positive mast cells are also present in lesional skin biopsies. One study found that horses affected by insect bite hypersensitivity had significantly more IgE-bearing cells in skin biopsies than healthy horses. [6]

Culicoides spp has been studied the most extensively and is considered a type I and IV hypersensitivity from the bites of Culicoides spp. A Th2 polarized response has been detected in horses that develop this type of hypersensitivity and appears to be linked to a decrease in IL-10 and higher IL-4 production compared to non-allergic horses.[7] More than 1000 species of Culicoides exist and depending on the geographic area, 30 - 40 of these can be active and feeding. Many different species may contribute to the clinical lesions of the disease with specific species having distinct feeding patterns on the body, feeding dorsally on the mane, tail, while others feed ventrally. [8] The main allergens are proteins found in the saliva of Culicoides. Further characterization of the salivary allergens were looked at in a study aimed to improve allergen immunotherapy. In this study numerous Culicoides allergens were produced as recombinant (r-) proteins using an allergen microarray. The results showed that Culicoides r-allergens in the IBH-affected horses had a significantly higher seropositivity than control horses for 25 r-allergens. Nine Culicoides r-allergens were major allergens for IBH with seven of them binding IgE in sera from > 70% of the IBH-affected horses. Combination of these top seven r-allergens could diagnose > 90% of IBH-affected horses with a specificity of > 95%.[8a] In addition, IgGa and IgGT but not IgGb were also found in affected IBH horses. Both the IgE and IgG were found in much higher levels in insect hypersensitive horses than healthy horses.[9] It has also been reported that healthy horses attract more biting midges than horses with insect hypersensitivity.[10] There is controversy regarding the cross reactivity of Culicoides allergens in horses with hypersensitivity There are many reports from all over the world supporting that species that are not native to a specific area can still create IDT reactivity whereas other studies show hypersensitivity requires a specific local indigenous. [8,12-22] There are also studies that show that different species of insects can exhibit cross reactivity with Culicoides with IgE cross-reactivity and co-sensitization against flies of genera Culicoides and Simulium.[23]

Epithelial barrier function has also been looked at in horses with IBH. One study examined skin immune responses by looking at transcriptome of lesional whole skin of IBH-horses. In the epidermis, genes involved in metabolism of epidermal lipids, pruritus development, as well as IL25, were significantly differentially expressed in lesional skin of IBH horses, suggesting an impairment of the epithelial barrier in IBH-affected horses that may act as a predisposing factor for IBH development.[23a]

In addition to Culicoides having specific feeding patterns other insect hypersensitivity lesions tend to reflect the insect feeding patterns. As mentioned, with Culicoides there are three main feeding patterns, dorsal, ventral and a combination of these. Dorsal pattern usually creates lesions over the face, pinnae, head, mane, withers and tail head. Ventral distribution can affect the intermandibular areas, thorax and abdomen, axillae, ventral midline and groin. *Haematobia irritans, Simulium* and some *Culicoides spp*. prefer to feed in these locations. *Haematobia irritans* (Horn flies) tends to favor a focal ventral

umbilicus location. The preferred feeding sites of *Stomoxys calcitrans* (Stable flies) and *Aedes* (Mosquitoes) are the caudal lateral aspects of both the font and hind limbs. The affected sites are characterized by intense self-trauma, crusting, alopecia and with more chronic lesions lichenification and scarring is seen.

Atopic dermatitis: Atopic dermatitis is an inherited predisposition to form sensitizing antibodies to environmental allergens such as the pollens of grasses, weeds, trees, molds and dust. Sensitizing antibodies (IgE) will bind to mast cells in the skin or respiratory tracts and ultimately end up creating a mast cell release of inflammatory mediators. To call the disease atopic dermatitis we must verify that horses make IgE in response to environmental allergens, that they have an imbalance between Th2 and Th1 cells, that they absorb allergens through the skin and that they have an impaired skin barrier. It is clear that horses make IgE and that allergen specific IgE can be detected using intradermal and/or serum testing. Based on what we know about mammalian IgE, we can assume that horses, like other allergic mammals, use the same immunologic mechanisms. While we lack strong evidence about pollens, molds, dusts or danders, there is good evidence that a Th2/Th1 imbalance is involved in horses with *Culicoides* hypersensitivity as described above and that this insect bite hypersensitivity shares many features with atopic dermatitis. As seen in other species it is likely that horses have a familial predisposition to atopic dermatitis and that many polymorphic genes are involved that influence the function of the innate and acquired immune responses as well as the structure and function of the skin barrier. Similar immunology also likely occurs in horses with an immune response that is skewed toward a T helper 2 response, IgE production is induced and a variety of cytokines, including IL-4, IL-5, IL-6, IL-13, and IL-31. An eosinophilic inflammatory infiltrate and pruritus are significant features of the atopic response in the skin of horses. There is a complex interplay between the immune system and the nervous system which promotes the sensation of itch. Th2 cytokines, particularly IL-31, directly stimulate itch by binding to their receptors on nerve fibers which is thought to be important in equine atopic dermatitis.[23b] IL-31 has been shown to be a potent stimulator of pruritus in horses and preliminary studies make IL-31 a reasonable target for future therapies in allergic horses. The role for IL-5 in IBH and a beneficial effect of vaccines targeting this cytokine have also been studied. On a cellular level, the skin lesions of IBH are characterized by massive eosinophil infiltration. IL-5 is a major regulator of eosinophils and in one study 17 of 19 horses developed IL-5 autoantibody titers and clinical improvement in scoring showed that 47% and 21% of vaccinated horses reached 50% and 75% improvement.[23c]

Other pruritogenic mediators likely play a part as well (histamine, proteases, substance P, opioids, neurotrophins and other neuroactive peptides). Secondary infections with Staphylococci and Malassezia yeast can also further aggravate the level of pruritus. Specific studies in the horse regarding Th-2 cells and allergies have been seen with COPD horses where increased numbers of T helper cells were found in bronchoalveolar lavage fluids, including increased numbers of lymphocytes expressing mRNA for IL-4 and IL-5 and reduced numbers of cells positive for IFN-gamma mRNA.[24] Heimann and colleagues used immunohistochemical staining to compare the distribution of CD4+, CD8+, and FoxP3+ T regulatory cells between normal horses and those with insect hypersensitivity. There were increased numbers of T cells in the affected horses, but

ratios of FoxP+ T cells/CD4+ were significantly lower in affected horses compared to normal horses.[25] Affected horses showed elevated mRNA levels for IL-13 in lesional and nonlesional skin and lower mRNA levels for IL-10 in lesional skin. These data could support the hypothesis that insect hypersensitivity in horses is associated with imbalances in the ratio of T helper 2 cytokines and those produced by regulatory T cells.

Barrier dysfunction is considered an integral part of the pathogenesis of atopic dermatitis. In fact, the skin barrier and the immune response are believed to be tightly linked. We know very little about the skin barrier of horses; one study established that some of the ultrastructural changes associated with barrier defects in humans and dogs were seen in the skin of two atopic horses.[26] This finding supports continued study into the barrier function of horses and whether barrier repair will become part of a multimodal approach to the management of atopic dermatitis in horses.

Many of the affected horses with atopic disease will present with similar findings as those seen with insect hypersensitivities. It is the authors' experience that it is extremely common to have both insect and atopic disease in the same horse. Pruritus with secondary lesions of alopecia, excoriations, lichenification and hyperpigmentation may be present on the ears, face, ventrum and legs. In some of these horses the pruritus may be accentuated by insect hypersensitivity. Urticarial lesions are also common in horses with atopic disease and in some cases pruritus and urticarial lesions can be seen in the same individual. Other ill-defined clinical signs that testimonials have been shown to be associated with atopic disease are asthma, laminitis and head tossing.

Head tossing: Head tossing is an interesting and often frustrating disorder. Allergies are but one of the proposed causes of this syndrome. Other reported causes include middle ear disorders, ear mites, rider ineptitude, auditory tube diverticulum (guttural pouch) mycosis, periapical dental osteitis, equine protozoal myeloencephalitis (EPM) and vasomotor rhinitis. Many cases exhibit seasonality, with could suggest that photoperiod and associated neurohumoral changes, ambient temperature and humidity, as well as exposure to allergens or other environmental triggers in headshaking syndrome. The author has only seen a limited number of these cases but has seen ASIT be effective in one case. There are other reports supporting allergy testing and allergen specific immunotherapy as well as many other treatments including antimicrobials, glucocorticoids (systemic and inhaled), antihistamines, gabapentin, alpha-2-agonists, fluphenazine, phenytoin and phenobarbital, melatonin, sodium cromoglycate eye drops fly control, acupuncture and cyproheptadine (0.3mg/kg BID).

Food intolerances or adverse food reactions: In general food intolerances and adverse food reactions in the horse are difficult to define and their true incidence is not known. Food intolerance or adverse food reactions in the horse is an allergic or idiosyncratic reaction to dietary grains, grasses, food additives or dietary supplements. Specific food groups that induce pruritus in the horse have been associated with wheat, oats, concentrates, barley, bran, alfalfa, and feed supplements. Allergic reactions to foods are thought to be caused by multiple immunologic mechanisms including type I, II, III and IV reactions. Other non-immunologic reactions can trigger mast cell release from

mechanisms that are unknown. Most food allergic horses would be considered with a non-seasonal pruritic disease or urticarial symptoms. Similar to other species allergy testing is not considered an accurate diagnostic test and elimination diet trials are needed.[26a] Pruritus and urticaria may be seen on parts of the body less likely to be affected by an insect hypersensitivity but not always and would include the lateral caudal thorax and flanks. Pruritus limited to the base of the tail would also make one concerned with food hypersensitivity. The author has seen one case with concurrent gastrointestinal symptoms in the form of diarrhea or soft stools.

Diagnosis: The diagnosis of an allergic skin disorder relies extensively on history and physical findings. Seasonal history is often seen initially, particularly with atopic disease and insect hypersensitivities. Food allergy typically has more of a year-round history. Ruling out other pruritic diseases is essential. Intradermal testing for insect and atopic dermatitis hypersensitivities adds minor diagnostic value and should be used primarily for allergen specific immunotherapy selection. Physical findings are listed as above. Dermatopathology can rule out other disease but aids little to the diagnosis (see below).

Dermatopathology: Dermatopathology may not be particularly rewarding in allergic horses as it is relatively nonspecific, but it can rule out other infectious, neoplastic and autoimmune disorders. Many allergic horses will exhibit mixed eosinophilic perivascular infiltrates with variable degrees of surface crusting, erosions and ulcerations. Other features seen could include spongiosis, exocytosis and patchy areas of hyper and parakeratosis. Focal areas of eosinophilic folliculitis and eosinophilic granulomas can be seen, more often in insect hypersensitivity.

Allergy Testing: Allergy testing in the form of intradermal testing (IDT) has been looked at extensively in the last decade. It has been primarily used for selection of allergens for ASIT in humans, dogs, cats, horses and other mammalian species, but some clinicians feel it may aid in diagnosis. IDT is an in vivo test that requires intradermal injection of allergies that in theory bind and bridge reaginic IgE antibodies on the surface of mast cells and result in mast cell degranulation resulting in wheal and flare reactions. An alternative to IDT is serum in vitro allergy testing (SIAT). Several laboratories offer equine SIVT in the United States including ALK-ACTT (Port Washington, NY), Heska Corporation (Fort Collins, CO 80525), Greer, Idexx Laboratories (Westbrook, Maine), Biomedical Laboratory (Austin, TX 78712), and Spectrum Laboratories (Phoenix, AZ 85281). To date the value of these tests and ASIT based on these tests has been controversial in the horse. Problems related to technique, non-specific binding, lack of standardization between labs, allergen preparation, and sample handlings are concerns. Most are using a polyclonal anti-IgE reagent; the specificity and the affinity of the reagents vary between labs. The in vitro tests are also expensive, often costing more than comparative canine assays. Lorch, et al found a sensitivity of 47.3% and a specificity of 81.7% with a positive predictive value of 68.7% and a negative predictive value of 64.7% in horses with atopic disease and horses without atopic disease using IDT as a criterion standard.[27] This study used three different allergen-specific assays and found that none produced the results similar to those obtained by IDT. Poor correlations between IDT and a ELISA using a monoclonal antibody specific for horse IgE, only 2/61 allergens

(Timothy and Quack) had substantial agreement between IDT and IgE ELISA.[28] In a recurrent airway obstructive disease (RAO) study looked at serological IgE ELISA test (Allercept), an in vitro sulfidoleukotriene (sLT) release assay (CAST) and intradermal testing (IDT). In all three tests the majority of the positive reactions was observed with the mite extracts (64%, 74% and 88% of all positive reactions, respectively) but none of the tests showed a significant difference between RAO-affected and control animals.[29] Another study evaluated and compared levels of allergen-specific IgE, using an ELISA method, in Icelandic horses, with and without IBH. The investigators also looked at patterns of allergen specific IgE to insects, pollens, molds and mites in those groups of horses and examined the clinical significance of employing two different cut-off levels for the ELISA. The use of two cut-off levels, 150 EA and 300 EA, did not eliminate the false positives. Horses with IBH had a higher number of positive reactions, than healthy controls and this was borderline significant (P=0.053). This study showed that serological testing with a high-affinity IgE receptor (FcepsilonR1alpha) is presently not suitable as a tool for establishing a diagnosis of IBH or equine atopy.[30] One study supported the value for SIVT where 27 horses that were reported to benefit from ASIT, 13 had their ASIT formulated based on the results of IDT, nine had their ASIT based on a serum test and five had both an IDT and a serum test. The success proportions of ASIT between skin tests, serum tests and both showed no statistical difference between the three groups. [31] These results likely reflect the impact of how well the clinician correlates positive reactions with the history of exposure to positive allergens and clinical symptoms. The author has also seen more positive ASIT outcomes with in vitro tests in the last few years and as in the canine results of SIVT should be used with history and physical exam for selection of allergens for immunotherapy.

Of course, IDT is not without its share of problems but has shown that allergic horses react more frequently to IDT then healthy horses.[32-34] It is not readily available for all practitioners and is often not financially practical for most practitioners to maintain the extracts and perform testing themselves. Specialists are not always available to do testing. Even when available there can be problems with false positive and false negative reactions. These can be minimized by the expertise of the allergist. But even with experts reading and grading of the testing can be discrepancies.[34a] The allergens to be utilized depend upon the geographical region, although many allergens are found worldwide. Most specialists utilize similar allergens to what is used in small animals with the addition of more insects and molds. Table I includes the test and allergen concentrations that the author utilizes at the Animal Dermatology Clinics of Southern California. There are concerns about testing concentrations for many allergens, particularly insect allergens. Work has been performed looking at the irritant threshold concentrations for many of the commonly used insect allergens.[35,36] Based on these studies the author has modified some of the insect testing concentrations, and these are reflected in Table I. Since molds are so ubiquitous and do not vary significantly between geographic locations, it can be difficult to choose which are important to test for. However moist and humid climates will have higher mold counts. Molds may also be more important in cases with airway disease. Once antigen selection has been made, they need to be obtained and prepared for testing. Allergenic extracts should be from a reputable supply company. The author uses aqueous allergens from Stallergenes Greer

Laboratories, Lenoir NC 28645 or ALK, Port Washington, NY 11050. Standard concentration of most pollen and mold allergens for testing is 1,000 PNU/ml (protein nitrogen units/ml) with insects having more variable testing concentrations (see above or Table I). Some allergens are also supplied in a weight to volume (W/V) and require alternative dilutions. Dilution schedules can be obtained from your allergen supply company. Solutions for skin testing should be made up fresh every 4 weeks to maintain appropriate potency.

To obtain optimal results with IDT the horse should be withdrawn from antihistamines for several days (5 -10) and oral glucocorticoids for 10-14 days prior to testing. Longer withdrawal periods may be needed if oral glucocorticoids have been used for extended periods of time or if long-acting injectable glucocorticoids have been used. A study evaluated intradermal testing and withdrawal times from hydroxyzine (after 500mg BID for 7 days) and dexamethasone (after 20mg/d for 7 days) in five horses without allergic symptoms before and after treatment with these drugs. Testing was repeated in 3 -4h, 7 days and 14 days after drug withdrawal. This study concluded that treatment of horses with dexamethasone or hydroxyzine for 7 days had no effect on testing results but did decrease IDT wheal diameters. Based on findings of this study, withdrawal times of 14 and 7 days for dexamethasone and hydroxyzine, respectively, prior to IDT can be recommended.[37] Skin testing usually requires sedation and shaving. The author has had good success utilizing xylazine hydrochloride intravenously. Phenothiazine tranquilizers should be avoided as they may inhibit IDT reactions. The best site for testing is the lateral cervical region above the jugular furrow, between the jaw and the shoulder. Stay below the mane as the skin is thicker and more difficult to inject in this location. The site should be clipped with a number 40 blade and sites ink marked for reference of antigen identification. Approximately .05 to .1cc of the antigen is injected intradermally. Injections should be made 2 cm apart to avoid overlapping of reactions and misinterpretation of results. Reactions should be evaluated at 15-30 minutes and if possible, at 45 minutes, 4-6 hours and at 24 to 48 hours. It may be impractical to do 24 - 48 hr reactions in many clinical situations. Owners can be advised to observe for late onset or delayed reactions (swellings) and can measure and report these via the phone. Reactions are subjectively interpreted as with small animals, scoring reactions 0, 1, 2 3, and 4. Grading is based upon size, demarcations, depth and turgor of the wheals compared to a positive control (histamine 1:100,000 dilution) and a negative control (saline). Reactions greater then 2/4 are considered positive.

As mentioned, there is no accurate in-vitro or in-vivo test for food allergies. The only accurate way to diagnose an adverse food reaction or intolerance is food avoidance. In small animals, we know that it now takes between 6-8 weeks or longer to document this however, the time limits have not been confirmed in the horse. The four weeks that is currently recommended may be too short and the author is currently recommending 6 weeks in the horse. It can be difficult to convince an owner to do an elimination diet in a horse. Selection of a protein source that is foreign or not commonly fed is recommended. The author has had success with timothy or barley if not routinely fed. In addition, elimination of unnecessary supplements, vitamins and other drugs should be discontinued for this time. At the end of the dietary trial the horse should be rechallanged with the

previous diet and/or supplements. Generally adding back one item every 5-7 days is recommended to determine which food group or protein is responsible.

Other treatment options: Treatment for allergic skin disorders is often best determined through appropriate rule outs and diagnostics. Avoidance or reduced allergen exposure is often the best method of management, however, many times it is impractical. Avoidance is also attempted when we suspect or can diagnose that we are dealing with an insect hypersensitivity. By knowing the specific type of insects involved with the hypersensitivity, you can focus on feeding sites on the horse to treat as well as where in the environment to focus on disruption of the insect's life cycle. For example, if *Tabanus, Chrysops, Haematobia irritans* and /or *Stomoxys calcitrans* is identified then the horse should be stable during the day as these flies are all daytime feeders. Moving affected horses away from stagnant water supplies can also be helpful. *Simulium* or black flies tend to favor moving water, such as nearby steams or washes. Using a fan in a box stall and using 32 fine meshing netting or screening can help protect against *Culicoides spp.* Reducing exposure is critical and may not necessarily be complete for control, but may aid along with other treatments. The author favors fly repellents with permethrin (1-5%%) as the primary insecticide and repellent. Concentrates of 10% permethrin can be purchased and diluted to the desired concentration. N,N-diethyl-m-toluamide (DEET) is also a good repellent but has no insecticide effects. However, one study did not show major statistically significant reduction of Culicoides 24h post treatment with a topical insecticide containing permethrin (3.6%).[20] (Fipronil a commonly used flea control product (Frontline, Merial) has also been used for insect control in hypersensitive horses. It is applied in the spray formulation at common fly feeding sites i.e., mane, tail head, legs and ventrum on a 2 – 3x a week basis. A useful insect eliminating device is the Mosquito Magnet ®. This kills mosquitoes, black flies, *Culicoides*, sand flies and other biting insects. It utilizes platinum beads to convert propane into carbon dioxide with a counter-flow technology that emits a plume of carbon dioxide, heat and octenol attractant and moisture from the inner attractant tube. The insects are attracted and do not fly across the plume and caught in a vacuum and dehydrate and die. Fly baits can also be helpful in reducing fly numbers. Older products utilized organophosphates and more recently imidacloprid. A recent product that utilizes a new class of insecticide, spinosyn, Elector Bait®, Elanco, has a delayed mode of action and flies die away from the bait and is extremely safe. Protective blanketing and fly shields can also be used successfully to protect against insect bites. Since dust mites can be recovered from horse blankets, for cases with dust mite allergies, washing blankets in hot water can also be of value. Dust mite and storage mite numbers can also be reduced in the stalls by using a borate based product (DUSTMITE, Ecology Works) that can keep mite levels suppressed for 3-4 months. For mold allergies, environmental mold control can be of some value and changing bedding types in stalls may be beneficial.

Besides avoidance, other treatment options rely more on topical (emollient, moisturizing and anti-pruritic) and systemic therapy (antihistamines, phosphodiesterase inhibitors, fatty acids, allergen specific immunotherapy and glucocorticoids). Many of the small animal topical products can be used that provide emollient, moisturizing and anti-pruritic effects. The author prefers products that contain colloidal oatmeal, essential fatty acids,

pramoxine and hydrocortisone. Additional antiseborrheic and antimicrobial agents can be used if secondary scaling, flaking and infections are present. There are several small animal products available through Bayer, Virbac, Vetoquinol, Ceva, Dechra and Vet Biotek. Topical glucocorticoids are also options for localized areas of pruritus. A product that is available in many countries outside of the US and may be a good choice for localized pruritus control in the horse is hydrocortisone aceponate (HCA; Cortavance®, Virbac SA, Carros, France), available as a 0.0584% spray formulation. As a nonhalogenated, di-ester topical glucocorticoid it is associated with better local and systemic tolerance compared to conventional topical glucocorticoids. One study looked at cutaneous atrophy in horses comparing several topical glucocorticoids (hydrocortisone, diflorasone diacetate, mometasone furoate and clobetasol propionate). The skin thinning effect of diflorasone diacetate, mometasone furoate, and clobetasol propionate was quite similar. Hydrocortisone showed only a weak skin thinning effect.[37a] In addition, the author feels that the lower limbs of horses are particular sensitive to this side effect and special care needs to be taken when using potent glucocorticoids in that location.

Antihistamines have classically been defined as chemicals that block the action of histamines at receptor sites. However, they may also have antipruritic effects and reduce urticarial reactions by stabilizing mast cells and having anti-serotonin properties. Although exact dosing and pharmacokinetics are lacking in the horse, many practitioners use these drugs. They typically have fewer side effects than glucocorticoids although they are not nearly as effective. One antihistamine used is pyrilamine maleate. It is given parenterally at a dose of 1 mg/kg. However, one study showed pyrilamine is poorly bioavailable orally (18%) and can be detected by sensitive enzyme-linked immunosorbent assay tests in urine for up to 1 week after a single administration. Despite this data suggests that the withdrawal time for pyrilamine after repeated oral administrations is likely to be at least 1 week or longer.[38] Clemastine and fenoxifenadine have a reported low bioavailability in horses of 3.4%[38a] and 2.6%[38b] respectively. In contrast, chlorpheniramine and cetirizine may be useful for atopic horses. Cetirizine has a high bioavailability in the horse, and at a dose of 0.4 mg/kg orally twice daily caused a significant inhibition of wheal formation after intradermal injection of histamine hydrochloride at 0.1 mg/mL.[38c] However, a randomized, placebo-controlled study evaluating its effect in a rather large number of horses with *Culicoides* hypersensitivity showed no difference in clinical scores between the treatment and placebo group.[38d] Hydroxyzine is considered one of the most frequently used antihistamines for horses.[38e] It is given at 1-2 mg/kg q 8-12 hours and is the authors favorite to use. Others used include doxepin is used at a dose of 0.75-1 mg/kg q 12 hours. chlorpheniramine at 0.1-0.5 mg/kg q 12 h, and diphenhydramine at 1-2 mg/kg q 8-12 h. Side effects are minimal and include light sedation, although occasional personality changes may be seen that may require reduction of dosages or discontinuation of the drug. The American Quarter Horse Association recommends a 10-day withdrawal prior to any shows or competition.

The author has used pentoxifylline (PTX), a methylxanthine derivative that is a potent inhibitor of PDE with strong anti-inflammatory properties, with anecdotal reports on the control of equine atopic dermatitis. It has been used in the equine for vascular diseases,

laminitis and for treatment of airway obstruction. The current dosing is ~15 mg/kg BID. Controversy exists on the pharmacokinetics of the drug in the horse and exact dosing is also not known. Results indicate PTX is rapidly absorbed and metabolized. Higher serum PTX concentrations, area under the curve, and bioavailability were observed after the first oral dose, compared with the last dose. Serum concentrations of both PTX and M1 reach serum concentrations considered to be therapeutic in humans and therapeutic in horses with endotoxemia. Some studies suggest increasing the dose rate to 30 mg/kg/day by either increasing the dosage with twice daily administration or by increasing the dosing frequency to three times daily.[39] It can be tried in atopic dermatitis and urticaria.

Essential fatty acid (EFA) supplementation has had increased use in the horse. It is aimed at modifying the arachidonic acid cascade and thereby reducing pruritus and urticaria associated with inflammatory mediators as a result of this cascade. The circulating fatty acid profiles and the acquisition and washout of fatty acids in response to n-3 supplementation were determined for horses in the one study. A fatty acid supplement high in eicosapentaenoic (EPA) and docosahexaenoic (DHA) acid was fed to deliver EPA plus DHA. These results indicate that the circulating fatty acid milieu in horses can be influenced through targeted supplementation.[40] In another study 14 horses with seasonal Culicoides hypersensitivity were given 20 grams daily of evening primrose oil and cold-water marine fish oil in an 80 to 20 ratio. The results were that 4 horses were no better, 5 horses were better, and 5 horses were much improved. In yet another study horses were fed 200 ml of linseed per day for a 6-week period and showed no significant change in pruritus or lesional surface areas. However, this time frame may have been too short to completely evaluate the potential benefits of n-3 fatty acids.[41] The author has seen limited success using similar fatty acid combinations but when used, utilizes a balanced high concentration of EFA found in Platinum Performance Equine (Platinum Performance, Inc.). EFA can be part of adjunctive therapy with other forms of therapy utilizing a multimodal approach to manage equine hypersensitivities.

Allergen Specific Immunotherapy (ASIT): There are now numerous studies demonstrating value for immunotherapy in both insect and environmental allergens. However, most studies have not been controlled and included only small numbers of horses. One study on *Culicoides* hypersensitivity evaluated ASIT in a double-blinded control fashion with poor results.[42] However, in another trial, all 10 horses with *Culicoides* hypersensitivity improved during immunotherapy, and seven of these horses deteriorated again after cessation of therapy. [14] Most authors reported a 60% to 71% good to excellent response to ASIT based on the results of intradermal testing. [15,32, 43-47] Reports evaluating the influence of multiple concurrent positive reactions to insects on the outcome of ASIT show conflicting results.[47-48] It may require a longer period of treatment to see positive results. In a placebo-controlled study by the author, 64% of the horses treated with vaccines showed a 50% or greater improvement compared to only 23% with placebo. These cases included both insect and pollen reactive horses.[46] In a large retrospective study 41 horses seen over a 17-year period were treated with ASIT and according to the owners surveyed, the overall response rate to ASIT was 84%.[31] This percentage of success as well as that in one other report of 92% in the management of equine urticaria,[47] appear higher compared to what is reported in other studies. It is likely that owner assessment was skewed by placebo effect or because of concurrent

medications. In the UCD study when the success of ASIT was evaluated as a sole therapy, 59% of the cases were well controlled with a further small percentage (9%) of horses being considered partial responders (i.e. concurrent medications administered with ASIT but glucocorticoids could be discontinued). This would give a total response rate of 69% (22 of 32 horses) putting the success rate closer to previous reports. Also of interest was that of the 30 owners who reported using antipruritic medications prior to beginning ASIT, 57% (17 of 30) reported being able to discontinue those medications with the addition of ASIT.[31] Another study looked at the benefits of ASIT over an extended period in a prospective clinical and immunological study. Nineteen horses received ASIT for up to 24 months. Horses were randomized to one of three treatment groups: ASIT based upon intradermal test (IDT) results (n = 7); allergen specific IgE results by ELISA (n = 6)]; or a combination based on IDT and ELISA results (n = 6). There was excellent agreement between allergen specific IgE concentrations (time 0) and both immediate and delayed IDT results), and between immediate IDT and IgG results. Specific concentration of serum IgE and IgG decreased significantly for 13% and 38% of allergens, respectively, that were included in ASIT.[48] These results suggest that ASIT provides significant clinical benefit and supports roles for both allergen specific IgE and IgG in the pathogenesis of equine AD. This data also suggests that the clinical benefits from ASIT may result from reduction of allergen specific IgE and IgG concentrations in serum. The most recent report on ASIT was based on a retrospective phone survey in 34 cases with urticaria and pruritus (n=11), non-pruritic urticaria (n=7), pruritus (n=6), RAO (n=9) and RAO with urticaria (n=1). In 33/34 on ASIT, the number of ASIT refills ranged from 0 to 11 (mean of 3.7 and median of 3) with intervals between refills ranging from 3 to 12 months. In 6/18 cases, 50–100% horses improved and remained on ASIT, 4/18 improved 75% or more and owner stopped ASIT with minimal return of clinical signs, 5/18 reported no improvement and discontinued ASIT. Three horses with RAO (one with concurrent urticaria) had improved. This study suggested benefits of ASIT and that a small number of affected horses may eventually be able to be weaned off ASIT without recurrence of clinical signs.[49] A recent larger retrospective study also evaluated atopic horses based on an owner survey. In this study, 14 owners of horses treated with ASIT returned the survey and 9/14 (64%) stated an alleviation of clinical signs. Eleven owners discontinued AIT after the first vial, recurrence of atopic dermatitis was seen in two of those horses and responded again to a repeat initiation of ASIT. Localized injection site reactions were occasionally seen in 6/14 patients (43%).[49a]

The ASIT technique is similar to what is used in small animals (see attached Equine ASIT schedule). Most horses required antigen booster injections at 7-to-14-day intervals, with volumes ranging from .5 to 1 cc. Shots are given subcutaneously over the lateral cervical area. Antigen reactions are uncommon, with swelling at injection sites being the most common, which generally resolve within 1-2 days. Angioedema and anaphylaxis is extremely rare in the author's experience. Oral ASIT can also be used in the horse. The author has only utilized this is a limited number of cases but is aware of 2 horses currently doing well on this. There one report with severe angioedema in a horse on oral ASIT, that when switched to injectable ASIT had no further reactions and was successfully managed. [49b]

Glucocorticoids: Systemic glucocorticoids are often required for short term relief and in some cases for longer term control. They are very frequently prescribed and certainly need to be used judiciously and in appropriate dosing and intervals. It is essential to make an accurate diagnosis before using glucocorticoid therapy to decide on the type, duration and the dose of therapy required. Therapeutic dosages are not determined for any glucocorticoid in any equine dermatoses and each case needs to be treated individually. Recommended dosages are merely guidelines to follow. The author relies primarily on two glucocorticoids in practice, prednisolone, and dexamethasone. Prednisone and prednisolone do not appear to be equal in the horse. Possible reasons why horses do not respond as well to oral prednisone are poor absorption, rapid excretion, failure of hepatic conversion to prednisolone or combination of all of these.[50] Depending on the severity of the case, dosages may need to be at the high or low end of anti-inflammatory levels to control most allergic hypersensitivity conditions. Most induction periods range from 7-14 days followed by a tapering period of 2-5 weeks and then a maintenance period that may be used for as short a time as a few months or indefinitely, depending on the severity of the case and the seasonality. Induction dosages for prednisolone are 0.5-1.5 mg/kg per day with maintenance dosages at 0.2-0.5 mg/kg every 48 hours. Some cases will be resistant to prednisolone and may respond to either injectable or oral dexamethasone. Often an initial loading dose of dexamethasone is needed at .02-0.1 mg/kg, which may be followed by an oral maintenance dosage of .01 -. 02 mg/kg every 48 to 72 hours. This regime is particularly helpful in more refractory cases. When using oral glucocorticoids, writing out the induction, tapering and maintenance dosages on a day-to-day basis is extremely helpful (see attached client handout schedule). Such a schedule allows safer administration at a "threshold dose" so that the case remains disease free.

The adverse reactions associated with glucocorticoid therapy are numerous, but the immune system, musculoskeletal system and gastrointestinal system are some of the more common organ systems that can be affected in horses. Clients should be warned about the increased risk for infections and the impact on wound healing. The development of gastric ulcers in horses with chronic glucocorticoid use has also been a topic of concern. However, a previous review of risk factors associated with the development of equine gastric ulcers did not find any correlation between previous corticosteroid administration and gastric ulceration.[51] One of the most controversial but poorly documented adverse reactions is the development of laminitis in horses treated with glucocorticoids. There are many proposed mechanisms on how glucocorticoids could cause laminitis. These include vasoconstriction and metabolic effects such as increased circulating insulin or glucose, decreased collagen production in the lamellar basement membrane and connective tissue, diminished keratin production in the hoof wall, and diminished growth from the coronary band.[52-55] There are cases of glucocorticoid-induced laminitis reported in the literature but there is poor scientific evidence documenting a direct correlation between the two. In a comprehensive evidence-based review of 13 publications with 40 cases of corticosteroid-induced laminitis, there was insufficient evidence to support such an association in healthy adult horses. However, there was weak evidence of an association between administration of

multiple doses of systemic corticosteroids and the onset of laminitis in adult horses with underlying endocrine disorders or severe systemic disease.[56]

TABLE I - Equine Skin Test

Animal Dermatology Clinic
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SOUTHERN CALIFORNIA EQUINE
INTRADERMAL ALLERGY TEST

Date:	Dr.:	Patient:	Client:

CONTROLS			**GRASSES**		
1. Saline			32. Bermuda *Cynodon dactylon*		
2. Histamine			33. Seven Grass Mix +		
INSECTS			34. Brome *Bromus inermis* +		
3. Mosquito *Culicidae*	1,000		35. P. Rye *Lolium perenne* +		
4. Mosquito *Culicidae*	250		36. Orchard *Dactylis Glomerata* +		
5. Deer Fly *Chrysops sp.*	1,000		37. Timothy *Phleum pratense* +		
6. Deer Fly *Chrysops sp.*	250		38. Alfalfa *Medicago sativa*		
7. Black Ant *Camponotus pennsylvanicus*	125		**TREES**		
8. Flea *Ctenocephalides canis/felis*	1,000w/v		39. Box Elder *Acer negundo*		
9. Flea *Ctenocephalides canis/felis*	400w/v		40. Palm *Arecastrum romanzoffianum*		
10. Storage Mite *Acarus Siro*			41. West. Juniper *Juniperus occidentalis*		
11. Storage Mite *Tyrophagus Putrescentiae*			42. Acacia *Acacia spp.*		
12. Culicoides *Variipennis*	1:1,000		43. Western Oak Mix *Quercus spp.*		
13. Culicoides *Variipennis*	1:10,000		44. Wester Walnut Mix *Juglans spp.*		
14. Horse Fly *Tabanus ssp.*	1,000		45. Olive *Olea europaea* +		
15. Horse Fly *Tabanus ssp.*	250		46. Melaleuca *Melaleuca quinquenervia*		
16. House Fly *Musca domestica*	1,000		47. Eucalyptus *Eucalyptus globulus*		
17. House Fly *Musca domestica*	250		48. Orange *Citrus sinensis*		
18. Moth *Lepidoptera*			49. CA Cottonwood *Populus fremontii* ++		
19. Dust Mite *D. Farinae*	250		50. Arroyo Willow *Salix lasiolepis* ++		
20. Dust Mite *D. Farinae*	62.5		51. White Mulberry *Morus alba*		
21. Caddisfly *Trichoptera*			52. Pepper Tree *Schinus spp.*		
22. Mayfly *Ephemeroptera*			53. Salt Cedar *Tamarix gallica*		
EPITHELIA			**WEEDS**		
23. Cat Dander *Felis catus*			54. Pigweed Mix *Amaranthus spp.*		
24. Feather *Chicken, Duck, Goose*			55. Lambs Quarter *Chenopodium album*		
25. Mouse *Mus musculus*			56. Russian Thistle *Salsola kali*		
26. Rat *Rattus norvegicus*			57. Firebrush *Kochia scoparia*		
27. Pyrethrum *Chrysanthemum cinerariifolium*			58. Western Ragweed *Ambrosia spp.*		
MOLDS			59. Sage Mix *Artemisia spp.*		
28. Curvularia *spicifera*			60. Dandelion *Taraxacum officinale*		
29. Fusarium Mix			61. Baccharis *Baccharis spp.*		
30. Mucor Mix			62. Mustard *Brassica spp.*		
31. Penicillium Mix			63. Dock/Sorrel Mix *Rumex spp.*		
			64. English Plantain *Plantago lanceolata*		
			65. Nettle *Urtica dioica*		

Scoring Legend
0 or left blank = no reaction
1 = low reaction
2 = moderate reaction
3 = strong reaction
4 = very strong reaction

If a "+" is beside a score = slightly larger than the score/number.

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Allergen Specific Immunotherapy (ASIT) Table 2

ASIT is one type of treatment for allergies in horses. The major benefit is its relative lack of side effects. Theoretically, it helps by creating tolerance, which allows your horse to be exposed to higher levels of allergens without developing symptoms such as hives, itching, rubbing, chewing, etc. ASIT is not always effective. Approximately 60% of the horses will be controlled. Of this 60% approximately 50% will be controlled without the use of other drugs. The additional 10% are helped, though they are not totally controlled and may require the use of other medications. The response to ASIT can be slow and gradual. Most horses do not respond until they have been on the injections for 3-6 months. Some may take as long as 9 months. Once they have responded, treatment will usually be needed for life. Your horse must be re-evaluated after 4-5 months and some adjustments may need to be made in his/her treatment. Side effects are rare. If swelling, itchiness, or hives appear within an hour of giving an injection, call the clinic. More serious side effects are very rare, but would include colic, diarrhea, respiratory difficulties, angioedema or collapse associated with the injection. Call our clinic or an emergency clinic at once if this occurs. More serious side effects generally occur within the first few months of therapy; therefore, you should give these injections when you will be with your horse for a minimum of 1-2 hours.

IMPORTANT

- **Antigens must be refrigerated**
- **Your horse must be re-evaluated during the 10-day injection intervals after 4- 6 weeks and again in 6 months**
- **It may take 6-9 months to show a response to antigens**
- **In most cases, antigen injections will be life-long**

Equine ASIT Schedule

DAY	DATE/SYMPTOMS†	AMOUNT	DAY	DATE/SYMPTOMS†	AMOUNT
VIAL #1			25		1.0cc
1		0.1cc	10 DAY INTERVALS (Recheck)‡		
3		0.2cc	35		1.0cc
5		0.3cc	45		1.0cc
7		0.4cc	55		1.0cc
9		0.6cc	14 DAY INTERVALS		
11		0.8cc	69		1.0cc
13		1.0cc	83		1.0cc
VIAL #2			97		1.0cc
15		0.2cc	111		1.0cc
17		0.3cc	20 DAY INTERVALS		
19		0.4cc	131		1.0cc
21		0.6cc	151		1.0cc
23		0.8cc	171	Recheck‡	1.0cc

†Record date and any increase or reduction in clinical signs

‡Call for a recheck appointment prior during 10-day cycle, some horses require volume and interval adjustments. A 6 month recheck should also be scheduled

DO NOT STOP INJECTIONS WITHOUT NOTIFYING YOUR Veterinarian

Infectious Diseases

Dermatophytosis and Malassezia Dermatitis: Dermatophytosis is a common, contagious superficial fungal infection of keratinized tissues. This usually includes the epidermis and hair and rarely the hooves. It is more common in younger horses or in debilitated or immunosuppressed horses. The incidence also increases in hot, humid climates or in horses kept in close contact with dark moist environments. More cases are seen in the fall and winter months. The most common species identified in the horse include *Trichophyton spp*. or *Microsporum spp. T. equinum var. equinum* and *var. autotrophicum* and *T mentagrophytes*. Most of these are zoophilic dermatophytes and transmission requires direct contact with infected animals or contact with infected hair or crusts in the environment. Hair loss with associated scale and crusting is the most common clinical sign. The alopecia is a result of hair shafts that are weakened by the fungus and then break or from deeper folliculitis. Lesions are usually multiple and vary in size and distribution. Typical lesions are 2-4mm in size and occur over the truncal, facial, head, axilla and chest locations. These sites are often where tack, blankets or saddles sit allowing the infection to occur more readily. Pruritus is not typically seen but can be present in some cases. Good review articles on equine dermatophytosis have been published.[57-60]

Malassezia dermatitis in the horse most commonly causes pruritus in the caudal intermammary area and tail head in mares but can create symptoms in male horses often over the prepucial area.[61] The exact species of Malassezia growing on horses needs further investigation. In one study, the *Malassezia spp* isolated were identified as *M. furfur, M. slooffiae, M. obtusa, M. globosa,* and *M restricta*.[62] Recent extensive reviews and guidelines have been published in small animals with equine and other food animal species reviewed. [63] Often there is a dry, greasy exudate in the intermammary folds. This often elicits a pruritic response. Treatment with 2% miconazole-chlorhexidine shampoos is usually effective.

Diagnosis is made by direct hair exams, cytology, fungal culture or biopsies. Optimally, microscopic examination of hyphae and macroconidia is needed for complete identification. For Malassezia cytology is adequate.

Treatment is not always necessary, and many cases are self-limiting, often disappearing within 1-3 months. Infected horses should be isolated, and all tack and grooming items should not be shared. Shaving the affected sites is of controversial value and may speed the healing process and reduce environmental contamination. Care should be taken when handling suspect or confirmed cases as this is a zoonotic disease and can create lesions in humans. Topical therapy in the form of shampoos and spray on rinses can be beneficial. The author prefers a poultry premise spray, 13.8% enilconizole, (Clinafarm EC, Schering Plough and Imaverol, Jannsen). This can be used as a 2% spray on 2-3 times a week to the affected sites. Other topicals that can be tried include 4 % lime sulfur, 2% chlorhexidine, 0.5% povidone-iodine, 0.5% sodium hypochlorite. Systemic therapy with griseofulvin has been used with a wide range of dosing 10 - 100mg/kg q 24h for 14-21 days. It can be tried in more resistant cases. It has limited availability and is a teratogenic drug and should be avoided in pregnant horses. Itraconazole and fluconazole have been

used to treat other mycotic infections in the horse such as coccidioidomycosis and aspergillosis at 2-5mg/kg q 12h and can also be tried but cost can be prohibitive.[64] 20% NaI may be given IV (250mg/500kg every 7 days for 1 -2 treatment courses. This also is contraindicated in pregnant mares as it may cause abortion. Other possible options include using Ethylenediamine dihydroiodide (EDDI) at a dose of 1 gram/day as used in cattle. Others have used EDDI as a feed additive formulation (Neogen Corporation, Lexington Kentucky) and dosed at 1 -2 mg/kg once to twice daily for first week then reduced to .5 to 1mg/d daily for next 2 – 3 weeks. Environmental treatment and disinfecting the tack, blankets, and grooming equipment with one of the above mentioned topicals should also be performed.

Staphylococcal Folliculitis: Although many bacterial skin infections in the horse appear as nodular or papular eruptions there are times when lesions can present in a more scaling and crusting pattern. Folliculitis due to staphylococcus species is common in the horse as it is in small animals and appears more frequently during the warmer summer months and in sites where tack, blankets or saddles rub or irritate the skin surfaces. The higher incidence in these locations and during the summer months has given such synonyms as summer rash or scabs, saddle sores or scabs or sweating eczema to describe this syndrome. Horses that are not properly rinsed and bathed after being worked may be predisposed. The author also believes that insect bites either aggravate or possible can be a source of vector inoculation of the staphylococcus in some cases. In addition to areas beneath the saddle, shoulders, lateral trunk and cervical areas, the distal limbs can be affected. The lower limbs and the pastern area can be significantly affected and can be a major differential when evaluating pastern dermatitis. *Staphylococcus pseudintermedius, Staphylococcus aureus, Staphylococcus hyicus* subsp. *hyicus*, and *Staphylococcus delphini* are recognized as the main staphylococcal species associated with bacterial pyoderma in horses, with *S. aureus* being common. In one study 128 strains of Staphylococcus from lesions, mostly from the skin were identified and compared with 29 strains isolated from the healthy skin. The pathogenic species *Staphylococcus aureus, S. intermedius* and *S. hyicus* were found almost exclusively in lesions. Other species including methicillin-resistant coagulase negative staphylococci (MR-CoNS) such as *S. xylosus , S lentus, S epidermidiis, S hemolyticus, S capitis and S. sciuri* can cause disease but are more frequently found on the healthy skin than in lesions.[65-70]

There are increasing worldwide reports of methicillin-resistant Staphylococcus aureus (MRSA) infection and colonization in horses and evidence that MRSA can be transmitted between horses and humans.[69ab] In horse farms, MRSA has been found circulating with prevalence ranging from 0.6% up to 4.7%, while higher prevalence (5.8%–12.0%) have been reported in horses admitted to veterinary hospitals.[70a] The majority of nosocomial infections in horses is associated with particular MRSA clonal lineages. Clonal lineages belonging to clonal complex (CC8) appear to be diminishing whereas MRSA attributed to CC398 is becoming increasingly more prevalent. Most of the CC398 isolates belong to a subpopulation which is particularly associated with equine hospitals as indicated by molecular typing.[70b] When emerging in equine clinics, MRSA from horses were also found as nasal colonizers in veterinary personnel. MRSA exhibiting the typing characteristics of MRSA from equine clinics are rare among MRSA from infections in

humans. Although rare so far epidemic MRSA from human hospitals (HA-MRSA, e.g., ST22, ST225) have been isolated from nosocomial infections in horses and need attention in further surveillance.[70] More details are available in the attached references. [65-81]

Risk factors for MRSA infections has also been looked at in horses. One study looked at the evolution of antibiotic resistance patterns before and after preventative pre and postoperative penicillin treatment. In this study staphylococci were isolated from skin and wound samples at different times during hospitalization. Hospitalization and preventive penicillin use were shown to act as selection agents for multi-drug-resistant commensal staphylococcal flora.[80] In other reports risk factors for MRSA colonization and infection showed administration of ceftiofur or aminoglycosides was associated with the acquisition of MRSA during hospitalization. And in other reports additional risk factors for community associated colonization included previous identification of colonized horses on the farm, antimicrobial administration within 30 days, admission to the neonatal intensive care unit, and admission to a service other than the surgical service. [66,68abcd]

Diagnosis is made on history and physical examination, cytology, culture and sensitivities and biopsies. Routine Diff Quik staining from intact papules or impression smears from crusted material is quite valuable. Skin biopsies often reveal bacteria in the surface crusts with occasional folliculitis identified.

Treatments with topical chlorhexidine shampoo is the author's favorite topical product. And whenever possible topical therapy should be the first line treatment. Some antibacterial ointments are highly effective in eradicating *S. aureus*. However, such topical antibiotics should be used when the size of the area to be treated is relatively small. When lesions are large, application of ointment is not practical and can be cost-prohibitive. The most frequently recommended topical antibiotics in equine literature are mupirocin and fusidic acid. In humans, several studies have reported emergence of fusidic acid and mupirocin resistance in countries in which they are widely used.[82] For these reasons, you should limit topical antibiotics for MRSA pyoderma, confirmed or highly suspected. Systemic antibiotics based on cytology or culture and sensitivity are also indicated in more severe cases. Trimethoprim sulfa is the main antibiotic used at 25mg/kg q 12h for 14 days but on occasion can exhibit resistance. Doxycycline can also be used at a dose of 10mg/kg q24hr. Many non-methicillin resistant cases will respond to procaine penicillin 22,000 – 44,000 IU q 12h for 14 days. Enrofloxacin (7.5 to 10 mg/kg BW, PO, q24h) should not be used as a first-line antibiotic to prevent antibiotic resistance. The fluoroquinolones are effective against bacteria susceptible to fewer antibiotics, such as *Pseudomonas* spp. It has been reported in humans and suggested in veterinary medicine, that exposure to fluoroquinolones may predispose to infection or carriage of MRSA.[68b] Ideally, fluoroquinolones should only be prescribed if the bacteria is susceptible to enrofloxacin based on culture and sensitivity. Other systemic antibiotics usually active against *S. aureus* are gentamicin (6.6 mg/kg, IM, SQ, IV, q24h), ceftiofur (2.2 mg/kg BW, IM, SQ, IV, q12 to 24h) or cephalexin (25 to 30 mg/kg BW, PO, q6 to q8h), chloramphenicol (35 to 50 mg/kg, PO, q6 to q8h) and rifampin (5mg/kg PO q 12h).[83] Care needs to be taken when using many of these antibiotics and resistance issues

have been seen even with more potent antibiotics such as chloramphenicol and rifampin.[84] Knowing bacterial susceptibility is ideal for choosing a systemic antibiotic, as well as the contraindications and reported side effects. Always prescribe the most effective and safest systemic antibiotics.

Basic hygiene practices should always be taken. Wash your hands thoroughly with soap and water or an disinfectant-based solution after working on infected or colonized horses. Wearing gloves can also reduce the risk of transmission of MRSA. Direct contact between a MRSA-positive horse and more susceptible humans is not recommended, as well as direct contact with other horses (avoid nose-to-nose contact). Equipment dedicated to an MRSA-positive horse should only be used for that horse. If the stall is to be occupied by another horse, the stall and equipment must be disinfected (washed with water and detergent, rinsed and dried, then disinfected with bleach or a conventional disinfectant) before giving access to another horse.

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