Pruritus in Horses: Causes and Control

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Pruritus often leads to self-inflicted trauma, alopecia, and moderate to severe secondary skin lesions resulting in horses which are visually disfigured and/or unsound for work. Because of the many possible diagnosis of a pruritic skin disease the history, especially the environment, seasonality, age of onset and progress of the disease, is very important in making a diagnosis. Time spent obtaining a detailed, complete history is rewarding and often equally as important as the examination and diagnostic tests.

A large percentage of horses affected with seasonal pruritic dermatitis are hypersensitive to the bites of insects. Any biting insect, including Culicoides spp. (No-see-ums, midges, punkies), stable flies (Stomoxys calcitrans), horn flies (Hematobia sp), black flies or "buffalo gnats" (Simulian spp.), horse and deer flies (Tabanidae and Chrysops spp.), mosquitoes (primarily Culex and Aedes spp.), can cause insect hypersensitivity dermatitis. "Chiggers" (Trombiculids) and ticks can also lead to hypersensitivity reactions. Insect bite induced hypersensitivities are characterized by intense pruritus which often leads to excoriation, extensive hair loss, secondary infections and chronically to hyperkeratosis and lichenification.

Several different clinical syndromes have been associated with hypersensitivities to different insects. Queensland or Sweet Itch, initiated by the bites of hematophagous Culicoides species, is classically characterized by a diffuse dorsal distribution with severe involvement of the mane and tail area. However, there are several species of Culicoides in different geographic areas and different species have different preferred areas of feeding. There are, for example, over 1000 species of Culicoides worldwide and as many as 30 or 40 species active in any one area. Work by Greiner et al in Florida has demonstrated multiple species feed on horses in different regions of Florida. These different species have variable feeding patterns with some species having a predilection for biting the
ventral aspect of the body instead of the dorsal. The only region generally not affected by *Culicoides* is the flank. An hereditary predisposition to develop hypersensitivity to *Culicoides spp.* has been shown in some breeds of horses and clinical evidence supports a familial tendency for hypersensitivity in many, if not all, breeds.

*Simulian* flies are blood suckers and bites are covered with small accumulations of dried blood. In mass attacks urticaria and angioedema may occur and can be associated with systemic signs of weakness with an increased pulse and respiratory rate, particularly in small young animals. Bites of *Simulian spp.* are most commonly associated with lesions on the head and within the inner pinnae, but in "swarm attacks" may bite any region of the horse. A generalized ventral distribution of lesions may also be caused by black flies.

Although *Culicoides spp* and *Simulian spp* are the most common and consequently the most studied there are a wide variety of gnats in certain regions of the country (such as the southeast) that occur in great numbers, can be found almost anytime of the day, and can lead to annoyance as well as irritating bites.

Bites of horn flies (*Haematobia irritans*) have been implicated in Ventral Midline Dermatitis, which is characterized by sharply demarcated focal lesions which often ooze serum. Horn flies prefer shade, and thus a few flies on a horse in the sunshine will be found on the ventral midline. In areas of heavy horn fly populations, these flies may be found everywhere on the body surface, including the body, legs and face. Horn flies breed only in cattle manure so most severe infestations are within a quarter of a mile of cattle farms. However, these flies may travel farther on wind currents, as they have been found on horses without nearby cattle.

Stable flies (*Stomoxys calcitrans*) are vicious biters (as opposed to house flies (*Musca spp*) that feed on body secretions). Initial lesions may consist of erythema, wheals, and raised nodules with a central crust. Exudative dermatitis of the legs due to reactions to extensive stable fly bites and/or urticaria and large nodules that are pruritic characterize hypersensitivities to stable flies.
Mosquito bites produce wheals and papules that are variably pruritic and are often painful. The lesions do not have a central crust as the bites of other insects. Most individual lesions resolve in 1-2 days, however larger persistent nodules may develop in certain individuals.

Chiggers and harvest mites (trombiculids) are free living and are found in undeveloped or overgrown fields and wooded areas. Adults are plant parasites, but the 6-legged larval form requires blood or tissue fluids for further development. Mites are non host specific and normally feed on small rodents. Infestations of horses may occur if horses are pastured or ridden in contaminated areas and are most prevalent in the summer and fall. Feeding larvae cause papules with 1-2mm crusts and can be intensely pruritic. Larvae remain on the host for 2-7 days before dropping off to molt and are often absent by the time the horse is presented for examination. The larvae are 0.2-0.4 mm long and yellow-orange to red in color and, if found, confirm the diagnosis.

Horses may develop hypersensitivities to ticks, either larvae ("seed ticks") or adults. Bites may induce individual large nodules with a central crust and/or ulcer or multiple bites may induce generalized edema, multiple nodules or ulcerated crusted areas. Very small larvae are easily overlooked even in massive infestations. Pruritus may be extreme, resulting in severe secondary lesions. Ticks are most commonly found on the legs, tail, head and ears.

Allergic diseases involve the interaction of three major factors: genetic constitution, exposure to allergens, and a dysregulation of the immune response determined by genetic predisposition and degree of exposure to allergens. However, other environmental factors such as infectious disease, contact with endotoxin and degree of infestation of endoparasites may also influence the prevalence of allergic diseases. Insect hypersensitivities are characterized by a Type I reaction. Type I hypersensitivity reactions develop in genetically programmed susceptible individuals and is mediated by IgE antibodies and some subtypes of IgG, which are produced by plasma cells near epithelial surfaces and specific to individual antigens. Specific antibody binds to the surface receptors of tissue mast cells and blood basophils, and, when cross linked by specific
antigen, causes degranulation of and release of inflammatory and vasoactive mediators from mast cells. These substances result in erythema, wheal formation and pruritus. The horse’s response to the intense pruritus leads to the clinical signs commonly present when the horse is presented for examination. The ability to respond to antigens by the production of IgE is largely inherited (atopy). Classic immediate hypersensitivity reactions are generally resolved by one hour. Late phase immediate hypersensitivity reactions are also mast cell dependent and occur 4-8 hours after challenge and may persist for 24 hours. Late phase Type I hypersensitivities are found in atopic individuals and in insect hypersensitivities. Insect hypersensitivities may also have a Type IV (delayed or cell mediated) reaction which is not associated with antibody development. The antigen is processed by Langerhan's cells which present a peptide fragments bound to the MCH class II antigens on the cell surface and presented to cells which become sensitized. Horses with IBH have been shown to have a strong Th2 type immune response with the relative “overproduction” of IL-4, IL-5, IL-6, IL-13 and IL-31. Lack of suppression of this response by T regulatory cells further enhances the immune response. A compromise in skin barrier function has been demonstrated in humans and dogs. Irregular or patchy components that form the tight junctions between epithelial cells allow for allergen entry, which is exacerbated by secondary skin infections. Furthermore, IL-31 binds directly to receptors on nerve fibers and promotes the sensation (along with other inflammatory mediators) of itch.

Many clinically affected horses positive on intradermal skin testing to one insect antigen are positive to other species as well. Queen and Baker studied a group of horses with a seasonally recurrent pruritic dermatitis with intradermal skin testing and passive cutaneous anaphylaxis tests and compared them to age and sex matched controls. Antigens of Culicoides, Stomoxys, Tabanidae, and Culex were used. All of the clinically affected horses responded most strongly within the first 4 hours after testing to Culicoides antigen. The horses also tested positive after 72 hours with the passive cutaneous anaphylaxis test. Half of the horses also had a delayed response (Type IV hypersensitivity reaction) to Culicoides antigen. Clinically affected horses were variably positive to the antigens of other
biting insects on both the intradermal and passive cutaneous anaphylaxis test. Their work has confirmed the clinical impression that horses with a hypersensitivity to one biting insect are often allergic to the bites of others.

Diagnosis

Diagnosis of insect bite hypersensitivity is made from the signalment (generally an adult individually affected animal, although related horses may be similarly affected), history, and response to avoidance (improve and exacerbate seasonally). The distribution of lesions on an affected horse is dependent on the biting characteristics of the insect responsible. Table 1 gives the general times of the day that different insects are active. Since there is still much to be learned about the identification and feeding habits of many of the insects implicated in allergic dermatoses, it may not be possible to identify the exact etiological agent. Intradermal skin testing is primarily used to direct treatment and is not used to make a diagnosis. Technical problems associated with testing included a lack of standardization of antigens and technique used. The saliva of some biting insects may also contain a histamine-like substance. Fifty percent or more of clinically normal horses in past reports have been found to respond positively to intradermal skin testing, although clinically normal horses generally respond to fewer antigens. Nevertheless, intradermal skin testing with currently available antigens on selected cases may provide identification of reactive antigens and guide part of therapeutic management.

Other allergies of the horse are atopic dermatitis, and/or food or contact allergies. Atopy is defined as a genetically programmed disease in which the patient becomes sensitized to environmental antigens that in a nonatopic animal creates no disease. Human and canine atopy has been proven to be genetically programmed and there is reasonable evidence that atopy of horses is also genetically determined. Atopic reactions are a Type I hypersensitivity to allergens (termed atopenes) that are absorbed percutaneously, inhaled or ingested. The hypersensitivity is not solely IgE and IgGd dependent and is a complex disorder of the immunologic system that results in a perturbation of many aspects of the immune response.
Environmental factors such as horses stabled most of the time early in life may be sensitized at an early age to molds found in barns; early high challenge with insect bites, parasitism, viral infections and vaccination with modified live virus vaccines, may all influence the onset and occurrence of clinical disease in atopic individuals. Atopic animals often exhibit clinical signs of disease much earlier than horses with acquired insect hypersensitivities. Atopic horses may present between 1 - 4 years of age, although they may be seen as old as 6 years. Clinical signs may be seasonal or persistent year round, dependent on the atopene. Many atopic horses' clinical signs will progress from seasonal to year round over time. The primary clinical signs are pruritus and urticaria that are generally symmetric. Regions of the body such as the face, ears, neck and legs may be affected or a more dorsal distribution of lesions on the mane, back and tail may occur. Atopic horses may also present with sterile eosinophilic folliculitis or tufted papules that become crusted and alopecic. It is important to remember that allergic reactions develop to common environmental antigens and that no new substances need be introduce to initiate the clinical disease. Secondary lesions caused by self inflicted trauma due to pruritus often result in more extensive lesions and secondary superficial infections. It is not unusual for atopic horses (particularly in the southeast) to have concurrent insect hypersensitivities, or, more rarely, a food allergy.

Diagnosis of atopy is made by exclusion of all other possibilities and is essentially a clinical diagnosis. Urticaria is not a diagnosis but a cutaneous reaction pattern that may be induced by a wide variety of causes, both immunologic and nonimmunologic. Rule outs for urticaria include drug and vaccine reactions, stinging and biting insects (such as wasps) and arachnids, infections, contacts, vasculitis, and cold, stress or exercise induced lesions. Intradermal skin testing is not used to make a definitive diagnosis but to identify allergens that can be avoided or used in allergen specific immunotherapy.

Food allergies are relatively rare and are poorly documented in the literature. Food allergies may be presented as persistent or recurrent urticaria or pruritus with accompanying secondary lesions. Food allergies are best diagnosed by limiting the diet to one forage for 4 weeks and then
adding back one foodstuff per week. Most authors prefer grass hay as high protein hays such as alfalfa or peanut hay may contain the inciting agent. Soy, a common ingredient in any commercial concentrate feed, is also a relatively common allergen. In the author's experience the most common offending feed is sweet feed or any other feed with molasses. An alternative to limiting the diet to just one forage is to eliminate molasses based feeds and/or commercial grain mixes for 3-4 weeks, then challenge the horse with the eliminated feed to determine the response to the feed.

Contact dermatitis is a Type IV hypersensitivity characterized clinically by hyperemia, papules, and vesicles and may appear like urticaria. It is generated by the percutaneous absorption of a protein which acts as a hapten and combines with a cutaneous protein. A T-cell response occurs against both hapten and protein. Once sensitized, lesions may be seen as early as 5-6 hours or as late as 24-72 hours after contact with the offending substance. Pruritus may be a component of the clinical response. Secondary skin changes associated with prolonged and/or repeated exposure include hyperpigmentation, lichenification, ulceration, and secondary pyoderma and mimic lesions associated with chronic insect allergies. Ingredients of many topical medications and insect repellents can produce contact allergies. Consequently care must be used in the selection and use of topical medications in other allergic dermatosis. Skin lesions due to agents producing irritation alone may also resemble contact allergies. Removal of the offending material as both diagnostic and curative and, although time consuming, offers the best solution.

**Intradermal Allergy Testing**

Antigens should be chosen from the environment of the horse, including insects, plants (grasses, weeds and trees), grains and forages. Both regional and seasonal variations in allergens exist, thus knowledge of the prevalence of plants and pollens help determine antigen selection. Use antigens in aqueous solutions as other diluents may be irritants to the horse and result in false positive reactions. Most published studies of intradermal allergy testing (IDT) in horses and the clinicians at the University of Georgia use allergens from Greer Laboratories. Most antigens are prepared in a 1:1000 weight/volume or 1000 Protein Nitrogen Units
(PNU)/ ml for injection. If the concentration of antigen is too high false positive reactions may occur. A wide variety of substances (alfalfa, grain mill dust, grain smuts, cottonseed, fireweed, yellowdock, Russian thistle, deer fly, black fly, horse fly, black ant, Rhizopus sp. and Canidida albicans) have been reported to be irritants in the horse and positive reactions must be interpreted with care. In the horse insect antigens and possibly other irritants may need to be further diluted to 250 or less PNU prior to use. However, it is best to test further diluted antigens with standard dilutions to help interpret the results. Separate insect antigens are superior to “mixed insects” as these contain too many proteins unimportant to clinical disease. Single insect antigens are available for most insects including Culicoides spp. At the present time all insect antigens for intradermal injection are made from the whole insect. These solutions contain many proteins which are not important to the clinical hypersensitivity to salivary antigens and may contribute to the irritation of these preparations when injected. Antigens should be stored in glass and not subjected to repeated freezing and thawing. Antigens that are too dilute or are outdated will result in false negative reactions as will injection of insufficient volume of the antigen solution.

False negative reactions may occur if the horse has received glucocorticoids, antihistamines or progestagens. There is no reliable information on withdrawal times of these medications prior to skin testing. Anecdotal withdrawal times, developed by individual clinicians, are: 3 weeks for oral or topically administered corticosteroids, 8 weeks for injectable corticosteroids, and 10 days for antihistamines and products and diets containing omega 3/omega 6 fatty acids. Phenothiazine tranquilizers and excessive excitement may also result in false negative reactions. Detomidine sedation is useful for testing excitable horses.

False positive reactions may occur if the allergen is innately irritant to the horse or if the allergen vial has become contaminated by bacteria or fungi. Poor intradermal injection technique, such as traumatic placement of the needle, use of a dull or burred needle, too large a volume of solution or air injected, may also result in false positive reactions. Occasionally horses may have "irritable skin" where large positive reactions are seen at all
injection sites, including the saline control. Despite the number of difficulties and inexact science of intradermal allergy testing, it remains the "gold standard" for identifying allergens for avoidance and allergen specific immunotherapy (AST), particularly for atopy.

It is important to remember that a positive ID skin test means a horse has skin sensitizing antibody; it does not mean clinical disease is present. Size of the reaction site does not necessarily correlate with the clinical importance of the allergen. Positive reactions are obtained in clinically normal horses. The frequency of positive reactions to molds and insects in clinically normal horses tends to increase with the age of the horse. Thus the results of ID test must be evaluated in concert with the historical and clinical findings.

**Serologic tests for circulating IgE**

In the RAST, or radioallergosorbent test, and ELISA, or enzyme linked immunosorbent assay, the antigen to be tested is attached to a solid substrate and are exposed to the patient's serum. Specific antibody binds to the antigen on the solid surface which is subsequently washed. The amount of bound IgE is determined by the addition of a labeled antiglobin with an indicator attached. In the liquid phase immunoenzymatic assay, a labeled allergen is mixed with the patient's serum. The resultant labeled allergen IgE complex is then bound by the label to a plastic well. This method purportedly decreases the incidence of false positive reactions due to nonspecific IgE binding. (This method however is not yet available for horse serum.) There is, however, poor correlation between the results obtained by serology and intradermal skin testing. Increased concentration of IgE in heavily parasitized animals may give false positive reactions. IgGd may also contribute to allergic dermatoses and is not detected by any of the serologic tests for IgE. Current methodology to overcome some of the limitations of serologic testing include prior absorbance of test serum for helminth associated and nonspecific IgE. Lorch et al investigated the correlation between ID testing and RAST and 2 ELISAs for circulating IgE concentrations in horses with atopic dermatitis, recurrent urticaria, and normal horses. Compared with the ID test, none of the 3 serum allergy tests detected allergen hypersensitivity
with any reliability. Consequently this author and others think IDT is a better test. A positive serologic test means a horse has circulating antibody; it does not mean clinical disease is present.

Control of atopy and insect-mediated hypersensitivities

Control of insect-mediated hypersensitivities by avoidance is the primary therapy. However complete avoidance can be difficult to impossible since several different insects with different feeding patterns may be involved. However, stabling of horses during peak feeding times of biting insects identified in clinical disease can substantially reduce the antigenic load. Stable flies, horse flies and deer flies are daytime feeders and prefer bright sunlight. Black (Simulian) flies are most active in the morning and evening, *Culiciodes* feed from dusk to dawn and mosquitoes are most active from dusk to 2 hours past sunset. Stabling horses in barns with clock operated mist sprayers of nonresidual pyrethrins may offer the best overall protection. Because some horses may develop a contactant or hypersensitivity response to repellants strategic use of fans to prevent insects from landing on the horse are advisable in stalls and loafing sheds. Screens used to exclude insects must have 60 squares per square inch of screen to exclude *Culicoides*. Permethrin products, which can be applied at less frequent intervals, may aid in protecting horses at pasture. Several spot on permethrin products with 44% - 64% permethrin are marketed specifically for horses. These products are applied to the poll, withers, tail head, and upper legs every 1-4 weeks (read labels). Topical permethrin sprays (look for > 2% concentration of permethrin or cypermethrin) can be used for other areas of the body on a daily basis. The use of petroleum jellies and oils as a mechanical barrier may decrease the occurrence of bites, especially when isolated areas of the body, such as the inside of the ears, are affected. Pyrethrin impregnated plastic mesh stable sheets are available and have been found useful by some owners as have "body suits" of light weight fly sheets and fine mesh.

The use of corticosteroids in topically applied creams is also helpful in localized reactions. Systemic corticosteroids remain as the best overall palliative therapy for generalized intense pruritus. Treatment should be initiated with 1-2 mg/kg prednisolone orally/day in the morning until the
pruritus and secondary skin trauma are under control. Then the dose may be decreased gradually to the least amount that will control the clinical signs. Every other day administration (0.5 mg/kg PO) is desirable for long term use. Prednisone may be used in some cases, but because of its limited and variable oral bioavailability not all horses will respond to its use. In severe nonresponsive cases dexamethasone at 0.05 -0.1 mg/kg parenterally or orally may interrupt the inflammatory response. Once the pruritus and skin lesion development is under control, prednisolone or prednisone may be substituted. A combination of the best avoidance control possible and oral every other day administration of prednisone or prednisolone often will provide adequate control.

Antihistamines, particularly hydroxyzine hydrochloride, can be used at a dose of 0.5-1.5 mg/kg every 8-12 hours orally. Hydroxyzine is more effective in controlling urticaria than pruritus, but is a useful part of the management strategy. Other antihistamines that may offer some benefit are: cetirizine (0.2-0.4 mg/kg BID); chlorpheniramine (0.25 mg/kg BID); diphenhydramine (0.75-1 mg/kg BID); pyrilamine maleate (1 mg/kg BID). Some authors also recommend tricyclic antidepressants doxepin hydrochloride (0.5 - 0.75 mg/kg BI or amitriptyline (1-2 mg/kg BID).

Hyposensitization against antigens identified by IDT may be helpful in some cases. The antigen solutions are made by the company supplying the antigens for intradermal testing. A series of injections with increasing concentrations of antigens (no more than 12-20 antigens) is given over several weeks until a final concentration is reached. Thereafter maintenance injections are given every 20-40 days. Improvement is gradual and generally recognized after 6- 12 months of treatment. The injection series should be timed such that the maintenance dose is achieved prior to the onset of the insect exposure or season. Experience with ASIT in horses has show 30-70 % of cases improve with therapy. Many horses with chronic insect hypersensitivities have a type IV (delayed) allergic response to insect bites as well as an immediate of type I response. Since delayed hypersensitivity is not immunoglobulin mediated, response to a hyposensitization regimen is less likely. It is important to remember that
hyposensitization begun during the middle of the insect season and height of the horse’s cutaneous response is very unlikely to improve the horse during the current season.

Atopic horses are the most likely to respond favorably to hyposensitization. Atopic horses improve at least 50% to 100% with partial improvement in 80% of atopic horses with concurrent insect allergies. A recent retrospective study reported that 84% of owners reported a good response in horses treated with immunotherapy. Of these, 93% of owners of horses that improved reported that their horses needed treatment with parenteral steroids prior to immunotherapy, and after 1 year of immunotherapy 59% were managed with immunotherapy alone. Systemic corticosteroids may be used as in insect hypersensitivities and, in cases of recurrent or persistent urticaria, hydroxyzine orally every 12 hours may control reoccurrence of hives. Hydroxyzine will not resolve existing urticaria. Doxipen, a tricyclic antidepressant that also has antihistaminic effects, has been used for atopy (0.6-1.2 mg/kg orally every 12 hours), however this author has no experience with it. Urticarial reactions may be associated with antigen exposure by systemic administration, inhalation, or ingestion. Definitive diagnosis of the etiologic agent is accomplished by removal and challenge. All possible antigens in the environment must be tested as hypersensitivity may develop to an antigen that has been present for a prolonged period of time. Recurrent urticaria can occur to common environmental antigens such as straw, blankets, tack as well as hay and grains.

**Differential Diagnoses**

Other dermatoses that may have also exhibit pruritus are onchocerciasis, oxyuriasis, dermatophytois, pemphigus foliaceus, pediculosis and chorioptic mange. Onchocerciasis, a hypersensitivity to the microfilaria of *Onchocerca cervicalis*, has a lesion distribution that can mimic Culicoides allergic dermatitis. Since *Culicoides spp.* are the major vector of Onchocerca, differentiation between the two can be difficult. Finding large numbers of microfilaria in the skin of clinically affected horses is helpful but not diagnostic, since the inflammatory reaction is often not associated with larvae on histological sectioning. Ivermectin and
moxidectin kill microfilaria in the skin within 14 days but do not eliminate adult *Onchocerca* in the ligamentum nuchae. Clinically, the incidence of onchocerciasis has decreased with the common and frequent use of ivermectin or moxidectin for internal parasite control. However, the recent emphasis on strategic use of anthelmintics for gastrointestinal parasite control may result in more frequent occurrence of this disease. Infestations with *Oxyuris equi* are characterized by tail rubbing with loss of tail hairs and excoriations of the underlying skin. Diagnosis is made by examining an acetate tape that has been applied to the anal region for the presence of *Oxyuris* ova. The "sticky" side of the tape is then placed on a glass slide that has been coated with mineral oil and examined under low power objective of a microscope for ova. Early infestations of dermatophytes may appear as localized wheals. Some infestations may be accompanied by a marked response to fungal products and have a more generalized edema, particularly on the legs which may be painful and/or pruritic. Pemphigus foliaceus may also be pruritic, particularly when horses are exposed to sunlight, as solar radiation exacerbates pruritus.

In cool weather lice and chorioptes mange are common conditions exhibiting pruritus. Chorioptes mites affect the lower limbs and are most frequently found on horses with large amounts of lower limb hair (draft breeds, Andalusians). Skin scrapings for diagnosis of chorioptes should be mixed with rotenone or other insecticide as the mites move very fast and may crawl off the slide before examination. Although sucking lice (*Haematopinus asini*) are controlled with avermectins, biting lice (*Bovicoli* (*Damalinia*) equi, *Wenedkiella equi equi*) and chorioptic mites feed on epithelial tissue debris and are poorly controlled by parenterally administered avermectins. These parasites are most effectively treated with topical agents such as lyme sulfur or permethrin. Persistence of parasite populations occur in low numbers often on asymptomatic animals, thus all in contact animals and common tack or grooming equipment should be treated.

Secondary skin infections with *Staphylococcus sp* or *Malassezia sp* may occur concurrently in atopic horses. It is well documented that atopic dogs are prone to both bacterial pyoderma and Malassezia infections. Staphylococcal infections may be an impetigo characterized by an
accumulation of neutrophils on the skin surface or may act as superantigens which enhance the activation of large numbers of T cells and contribute to the inflammatory reaction in the skin. In this case small groups of staphylococci may be seen adherent to keratinocytes without the presence of neutrophils. *Malassezia sp* are considered to be commensal surface organisms in dogs but in one study were not demonstrated on normal horses. Clinical infections of *Malassezia* infections are characterized by greasy to waxy foul smelling variably pruritic dermatitis in the axillae, groin, udder and prepuce. Superficial overgrowth of staphylococcal organisms and *Malassezia* infections may be treated topically with shampoos and antibiotics and antifungal agents respectively. (Equisheild CK Shampoo®, Kinetic Vet) Staphylococcal impetigo responds best to systemic antimicrobial treatment.

In all cases of pruritic dermatitis managerial procedures to decrease exposure to exciting agents as well as symptomatic therapy to reduce pruritus is warranted. All avenues available to decrease exposure to insects should be fully implemented. Heat, humidity and solar radiation exacerbate pruritus thus the provision of shade and wind currents by fans can provide relief. Simple feeds and whole grains are better than mixed multiple grain sweet feeds. Cold water rinses and shampoos can rehydrate dry skin and reduce the amount of topical allergens on the skin. Colloidal oatmeal, pramoxine (a topical anesthetic) and 1% hydrocortisone shampoos (hydrocortisone is not absorbed) may reduce pruritus and minimize or reduce the amount of systemic antipruritics needed. A complete and detailed investigation into the etiology of the disease should occur simultaneously with general symptomatic care. The client must understand that hypersensitivities and atopy are life long and the horse will need continuous management and or therapy. Often a patient may be symptom free with low exposure to inciting antigens and symptomatic as the antigen "load" increases. Antigen exposure is additive, thus comprehensive management is needed to best control clinical disease.

\textsuperscript{a} Greiner EC. Personal communication, 1986

\textsuperscript{b} White, SL. Unpublished data
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Stepnik CT et al. Vet Dermatol 2012; 1: 29-35 TABLE 1

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