**Diseases of the skin**

**The major functions of the skin are**:

* To maintain a normal body temperature
* To maintain a normal fluid and electrolyte balance within the animal
* To act as a sensory organ perceiving those features of the environment which are important to the subject's survival.

**Diseases of the skin :-** Diseases of the skin may be:-

1. Primary

2- Secondary in origin.

**Primary skin disease** the lesions are restricted initially to the skin although they may subsequently spread from the skin to involve other organs.

**Secondary** :- cutaneous lesions may be secondary to disease originating in other organs.

Differentiation between primary and secondary skin diseases should be attempted by seeking evidence that organs other than the skin are affected. If there is no such evidence produced during a complete clinical examination of the patient, it is reasonable to assume that the disease is primary.

Even if involvement of other organs is diagnosed it is still necessary to determine whether the involvement constitutes the primary state or whether it has developed secondarily to the skin disease . This decision can be based on the chronology of the signs, elicited by careful history taking, and a detailed knowledge of the individual diseases likely to be encountered.

When a careful clinical examination has been made and an accurate history taken it is then necessary to make a careful examination of the skin itself, using the proper technique of examination, especially (and essentially) histopathological examination of a biopsy specimen. It is then possible to determine the basic defect, whether it be inflammatory, degenerative or dysfunctional, and thus to define the type of lesion present.

**Clinical sfgbvc igns and special examination**

* A general clinical examination is followed by
* a special examination of the skin and must include inspection and, in most cases, palpation.
* Additional information can be obtained by

1-taking swabs for bacteriological examinations,

2-scrapings for examination for dermatophytes and metazoan parasites,

3-and biopsy for histopathological examination. Biopsy material should include abnormal, marginal, and normal skin. Artifacts are common in biopsy specimens, including non representative sampling, crushing the specimen by forceps or hemostat, and inadequate fixation.

**Descriptions of lesions should include:-** size, depth to which they penetrate, distribution on the body and size of the area covered. Abnormalities of sebaceous and sweat secretion, changes in the hair or wool coat and alterations in color of the skin should be noted, as should the presence or absence of pain or pruritus and the manifestations of skin

disease and the common lesions defined below:

**Lesions**

An accurate definition of the lesions, is an essential part of a skin patient's clinical record. The table makes a primary differentiation into

discrete and diffuse lesions in terms of size, e.g. they may be limited diffuse lesions or extensive localized ones.

|  |  |
| --- | --- |
| **Diffuse lesion**  Scales Dry,  Excoriations  Fissures  Dry gangrene  Early, moist gangrene  Keratosis  Acanthosis  Hyperkeratosis  Para keratosis  Eczema  Hypermelanosis  Hypomelanosis | **Discrete lesions**  Vesicle, bleb, buIla, blister  Pustule  Wheal  Papules (pimples)  Nodules, nodes  Plaque  Acne  Comedo  Impetigo  Scab (or crust)  Macule (patch) |

**Abnormal coloration**

This parameter includes jaundice, pallor and erythema, and these are best seen in the oral or vaginal mucosa or in the conjunctiva. In animals they are rarely visible in light-colored skins.

\*Red-purple discoloration of the skin of septicemic,

\*Early erythema is a common finding where more definite skin lesions

are to develop, as in early photosensitization.

\*The blue coloration of early gangrene (e.g. of the udder and teat skin

in the early stages of peracute bovine mastitis associated with Staphylococcus aureus) is characterized by coldness and loss of elasticity.

* Hypopigmentation of the skin:- may be general. As

1. in albino, pseudoalbino and lethal white animals.
2. Local patches of hypopigmentation are characteristic of vitiligo

and leukoderma.

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**Pruritus**

Pruritus or itching is the sensation that gives rise to scratching Hyperesthesia is increased sensitivity to normal stimuli

" Paresthesia is perverted sensation, a subjective sensation, and not diagnosed in animals.

All sensations that give rise to rubbing or scratching are therefore included with pruritus, more properly defined as scratching.

Pruritus can arise:-

from \*Peripheral or \*Central stimulation.

**peripheral in origin** :-

It is a primary cutaneous sensation like heat, cold, pain and touch; it differs from pain because it is purely epidermal. whereas pain can still be felt in areas o f skin denuded of epidermis. Thus itching does not occur in the center of deep ulcerations nor in very superficial lesions, such as those of ringworm, where only the hair fibers Skin and keratinized epithelium are involved.

Itching can be elicited over the entire skin surface but is most severe at the mucocutaneous junctions. Common causes include the following.

**cattle**

* Q Sarcoptic and chorioptic mange
* Aujeszky's disease
* Nervous acetonemia
* Lice infestation.

**Sheep**

* Lice, mange, ked, blowfly and itch mite infestations
* Scrapie.

**Horses**

* Chorioptic mange on the legs
* Queensland (sweet) itch along the dorsum of the body
* Lice infestation
* Perianal pruritus due to Oxyuris equi infestation.

**All species**

* The early stages of photosensitive dermatitis
* Urticarial wheals in an allergic reaction
* Licking syndromes' such as occur in cattle on copper-deficient diets are accompanied by pica and the licking of others as well as themselves. They are examples of depraved appetites developed in response to nutritional deficiency and are not a response to pruritus.
* **Itching of central origin derives in** the main from the scratch center below the acoustic nucleus in the medulla. It may have a structural basis, as in scrapie and pseudorabies, or it may be functional in origin, as in the nervous form of acetonemia. The only lesions observed are those of a traumatic dermatitis with removal of the superficial layers to a variable depth, breakage or removal of the hairs and a distribution of lesions in places where the animal can bite or rub easily.

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**Secretion abnormalities of skin glands**

The activity of the sweat glands is controlled by the sympathetic nervous system and is for the most part a reflection of body temperature. Excitement and pain may cause sweating due to cerebral cortical activity. A generalized form of hyperhidrosis, apparently inherited, has been recorded in Shorthorn calves. Local areas of increased or decreased sweating may arise from peripheral nerve lesions or obstruction of Principles of treatment of diseases of the skin sweat gland ducts. A generalized anhidrosis is recorded in horses and occasionally in cattle. Excess secretion of sebum by sebaceous glands causes oiliness of the skin or seborrhea but its pathogenesis is poorly understood.

**Abnormalities of wool and hair fibers**

alopecia or hypotrichosis :-Deficiency of hair or wool in comparison to the normal pilosity of the skin area .

Hirsutism:- abnormal hairiness, manifested by a long, shaggy, usually curly, coat is most common in aged ponies with adenomas of the pars intermedia of the pituitary gland.

The character of the fiber may also vary with variations in the internal environment.For example, in copper deficiency the crimp of fine wool fibers is lost and the wool becomes straight and 'steely'. Alternation in coat color, achromotrichia, may be generalized or segmental along the fiber.

**Principles of treatment of diseases of the skin**

Primary treatment

Primary treatment commences with

* removal of hair coat and debris to enable topical applications to come into contact with the causative agent.
* Accurate diagnosis of the cause must precede the selection of any topical or systemic treatment.
* In bacterial diseases sensitivity tests on cultures of the organism are advisable.

Specific skin diseases due to bacteria, fungi and metazoan parasites are reasonably amenable to treatment with the appropriate specific treatment.

Bacterial resistance to antimicrobials used in veterinary dermatological practice is a concern. The broad application of antimicrobials for various therapeutic and non therapeutic purposes has accelerated the spread of pre- existing resistance genes and led to the apparent development of mechanisms by which resistance genes are spread, not only within a bacterial species, but also between bacterial species. Every veterinarian must be careful in using of antimicrobials in order to minimize the resistance problem.

Removal of the causative agent in allergic diseases and photosensitization , may be impossible and symptomatic treatment may be the only practicable solution. In many cases, too, the primary disease may be confounded by the presence of a secondary agent, which can lead to confusion in diagnosis. Treatment may be unsuccessful if both agents are not treated.

SUPPORTIVE TREATMENT

Supportive treatment includes prevention of secondary infection by the use of bacteriostatic ointments or dressings and the prevention of further damage from scratching.

* Effective treatment of pruritus depends upon the reduction of central perception of itch sensations by the use of ataractic, sedative or narcotic drugs administered systemically or on successful restraint of the mediator between the lesion and the sensory end organ. In the absence of accurate knowledge of the pathogenesis of pain it is usual to resort to local anesthetic agents, which are short lived in their activity, and corticosteroids, which are longer acting and effective, provided that vascular engorgement is part of the pruritus-stimulating mechanism,
* When large areas of skin are involved it is important to prevent the absorption of toxic products by continuous irrigation or the application of absorptive dressings. Losses of fluid and electrolytes should be made good by the parenteral administration of isotonic fluids containing the necessary electrolytes
* Ensure an adequate dietary intake of protein, particularly sulfur-containing amino acids to facilitate the repair of skin tissues )

**Diseases of the epidermis and dermis**

**Pityriasis**

**Primary pityriasis**, excessive bran-like scales on the skin, characterized by overproduction of keratinized epithelial cells, can be caused by:

* Hypovitaminosis A
* Nutritional deficiency of B vitamins, especially of riboflavin and nicotinic acid, in pigs, or linolenic acid, and probably other essential unsaturated fatty acids
* Poisoning by iodine
* Pityriasis rosea occurs in humans and pigs and the etiology is unknown. The literature for and against an infectious etiology has been reviewed.

**Secondary pityriasis**, characterized by excessive desquamation of epithelial cells is usually associated with:

* Scratching in flea, louse and mange infestations
* Keratolytic infection, e.g. with ringworm fungus.

Pityriasis scales are accumulations of keratinized epithelial cells, sometimes softened and made greasy by the exudation of serum or sebum. Overproduction, when it occurs, begins around the orifices of the hair follicles and spreads to the surrounding stratum corneum.

Primary pityriasis scales are superficial, accumulate where the coat is long, and are usually associated with a dry, lusterless coat. Itching or other skin lesions are not features. Secondary pityriasis is usually accompanied by the lesions of the primary disease. Pityriasis is identified by the absence of parasites and fungi from skin scrapings.' \

! **Differential diagnosis**

* + Hyperkeratosis
  + Parakeratosis

**TREATMENT**

1-Primary treatment requires correction of the primary cause.

2-Supportive treatment commences with a thorough washing, followed by

alternating applications of a bland, emollient ointment and an alcoholic lotion. Salicylic acid is frequently incorporated into a lotion or ointment with a lanolin base.

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**HYPERKERATOSIS**

Epithelial cells accumulate on the skin as a result of excessive keratinization of epithelial cells and intercellular bridges, interference with normal cell division in the granular layer of the epidermis and hypertrophy of the stratum corneum.

Lesions may be:-

**A- local** at pressure points, e.g. elbows, when animals lie habitually on hard surfaces.

**B-Generalized** hyperkeratosis may be caused by:

* Poisoning with highly chlorinated naphthalene compounds
* Chronic arsenic poisoning
* Inherited congenital ichthyosis
* Inherited dyserythropoiesis- dyskeratosis.

The skin is dry, scaly, thicker than normal, usually corrugated, hairless and fissured in a grid like pattern. Secondary infection of deep fissures may occur if the area is continually wet. However, the lesion is usually dry and the hyperkeratotic material can be removed, leaving the underlying skin intact.

Confirmation of the diagnosis is by the demonstration of the characteristically thickened stratum corneum in a biopsy section, which also serves to differentiate the condition from parakeratosis and inherited ichthyosis.

Primary treatment depends on correction of the cause. Supportive treatment is by the application of a keratolytic agent (e.g. salicylic acid ointment) .

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**Parakeratosis**

Parakeratosis, a skin condition characterized by incomplete keratinization of epithelial cells, can be: Caused by

* nonspecific chronic inflammation of cellular epidermis
* Associated with dietary deficiency of zinc
* Part of an inherited disease.

The initial lesion comprises edema of the . prickle cell layer, dilatation of the intercellular lymphatics, and leukocyte infiltration. Imperfect keratinization of epithelial cells at the granular layer of the epidermis follows, and the horn cells produced are sticky and soft, retain their nuclei and stick together to form large masses, which stay fixed to the underlying

tissues or are shed as thick scales.

The lesions may be extensive and diffuse but are often confined to the flexor aspects of joints (referred to historically in horses as mallenders and sallenders) . Initially the skin is reddened, followed by thickening and gray discoloration. Large, soft scales accumulate, are often held in place by hairs and usually crack and fissure, and their removal leaves a raw, red

surface. Hyperkeratosis scales are thin, dry and accompany an intact, normal skin.

Confirmation of a diagnosis of parakeratosis is by the identification of imperfect keratinization in a histopathological examination of a biopsy or a skin section at necropsy.

**Differenial diagnosisIF**FERENTIAL DIAGNOSIS

• Hyperkeratosis

• Pachyderma

• Ringworm

• Inherited ichthyosis

• In herited Adema disease in cattle

• Inherited epidermal dysplasia

**TREATMENT**

**Primary treatment** requires correction of any nutritional deficiency.

Supportive treatment includes removal of the crusts by the use of keratolytic agent (e.g. salicylic acid ointment) or by vigorous scrubbing with soapy water, followed by application of an astringent (e.g. white lotion paste), which must be applied frequently and for some time after the lesions have disappeared.

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**Impetigo**

A superficial eruption of thin -walled, small vesicles, surrounded by a zone of erythema, that develop into pustules, then rupture to form scabs.

In humans, impetigo is specifically a streptococcal infection but lesions are often invaded secondarily by staphylococci. In animals the main organism found is usually a staphylococcus. The causative organism appears to gain entry through minor abrasions, with spread resulting from rupture of lesions causing contamination of surrounding skin and the development of secondary lesions. Spread from animal to animal occurs readily. The only specific examples of impetigo in cows are: Udder impetigo of cows

Small (3-6 mm) vesicles appear chiefly on the relatively hairless parts of the body and do not become confluent. In the early stages each vesicle is surrounded by a narrow zone of erythema. No irritation is evident. Vesicle rupture occurs readily but some persist as yellow scabs. Involvement of hair follicles is common and leads to the development of acne and deeper, more extensive lesions. Individual lesions heal rapidly in about a week but successive crops of vesicles may prolong the duration of the disease.

Confirmation of the diagnosis is by culture of vesicular fluid and identification of the causative bacterium and its sensitivity.

**Differential diagnosis**

• Cowpox, in which the lesions occur almost exclusively on the teats and pass through the characteristic stages of pox

• Pseudocowpox, in which lesions are characteristic and also restricted in occurrence to the teats.

**Treatment**

1-Primary treatment with antibiotic topically is usually all that is required

because individual lesions heal so rapidly.

2- Supportive treatment is aimed at preventing the occurrence of secondary lesions and spread of the disease to other animals. Twice daily bathing with an Diseases of the epidermis and dermis efficient germicidal skin wash is usually adequate.

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**URTICARIA**

An allergic condition characterized by cutaneous wheals. It is most common in horses.

**ETIOLOGY:**Primary urticaria results directly from the effect of the pathogen, e.g.:

1-Insect stings

2-Contact with stinging plants

3-Ingestion of unusual food, with the allergen, usually a protein

4-Occasionally an unusual feed item, e.g. garlic to a horse

5-After a recent change of diet

6-Administration of a particular drug, e.g. penicillin; or anesthetic agent

7-Allergic reaction in cattle 8 days following vaccination for foot-and mouth diseases

8-Death of warble fly larvae in tissue

9-Milk allergy when Jersey cows are dried off

10-Transfusion reaction.

Secondary urticaria occurs as part of a syndrome, e.g.:

* Respiratory tract infections in horses, including strangles and the upper respiratory tract viral infections

**Pathogenesis**

The lesions are characteristic of an allergic reaction. There is degranulation of mast cells followed by liberation of chemical mediators inflammation, resulting in the subsequent development of dermal edema.

A primary dilatation of capillaries causes cutaneous erythema. Exudation from the damaged capillary walls causes local edema in the dermis and a wheal develops.

Only the dermis, and sometimes the epidermis, is involved. In extreme cases the wheals may expand to become seromas, when they may ulcerate and discharge. The lesions of urticaria usually resolve in 12-24 hours but in recurrent urticaria an affected horse may have persistent and chronic eruption of lesions over a period of days or months.

**CLINICAL FINDINGS**

Wheals, mostly circular, well delineated, steep-sided, easily visible elevations in the skin, appear very rapidly and often in large numbers, commencing usually on the neck but being most numerous on the body. They vary from 0.5-5 cm in diameter, with a flat top, and are tense to the touch. There is often no itching, except with plant or insect stings, nor discontinuity of the epithelial surface, exudation or weeping. Pallor of the skin in wheals can be observed only in unpigmented skin.

Other allergic phenomena, including diarrhea and slight fever, may accompany the eruption. The onset of the lesions is acute to peracute with the wheals developing within minutes to hours after exposure to the triggering agent. When associated with severe adverse systemic responses, including apnea, respiratory arrest, atrial fibrillation, cardiac arrest or sudden death, the case qualifies as one of anaphylaxis.

Subsidence of the wheals within 24-48 hours is usual but they may persist for 3-4 days because of the appearance of fresh lesions. Urticaria lasting 8 weeks or longer is classified as chronic or recurrent Urticaria, which may require testing for atopic disease using intradermal skin testing and serum testing for antigen-specific IgE.

Adverse reactions in dairy cattle following annual vaccination for foot and- mouth disease are characterized by wheals (3-20 mm in diameter) covering most of the body, followed by exudative and necrotic dermatitis. The affected areas become hairless and the wheals exude serum and become scabbed over. Edema of the legs is common and vesicles occur on the teats. The lesions appear 8-12 weeks postvaccination and may persist for 3-5 weeks. Loss of body weight and lymphadenopathy also occur. Pruritis, depression and a drop in milk yield are common.

**CLINICAL PATHOLOGY (Diagnosis)**

Intradermal skin tests to detect the presence of hypersensitivity are of little value because many normal horses, as well as those with urticaria, will respond positively to injected or topically applied allergens. Also, reactions usually occur within the first 24 hours after the injection, but the interval is very erratic. The duration of the reaction also varies a great deal. Intradermal tests in horses without atopy and horses with atopic dermatitis or recurrent urticaria using environmental allergens indicate a greater number of positive reactions for intradermal tests in horses with atopic dermatitis or recurrent urticaria, compared with horses without atopy. This provides evidence of type-1 IgE-mediated hypersensitivity for these diseases.

Biopsies show that tissue histamine levels are increased and there is a local accumulation of eosinophils. Blood histamine levels and eosinophil counts may also show transient elevation.

**Differential diagnosis**

The differential diagnosis list is limited to angioedema, but in urticaria the lesions can be palpated in the skin itself. Angioedema involves the subcutaneous tissue rather than the skin and the lesions are much larger and more diffuse. The two conditions may appear in the one animal at the one time.

**TREATMENT**

**Primary treatment**

A change of diet and environment, especially exposure to the causal insects or plants, is standard practice. Spontaneous recovery is common.

**Supportive treatment**

* Corticosteroids, antihistamines, or epinephrine by parenteral injection provide the best and most rational treatment, especially in the relief of the pruritus.
* The local application of cooling astringent lotions such as calamine or white lotion or a dilute solution of sodium bicarbonate is favored.
* parenteral injections of calcium salts are used with apparently good results.
* Long-term medical management of persistent urticaria involves the administration of corticosteroids and or antihistamines. Oral administration or prednisone or prednisolone at the lowest possible dose on alternate days is the method of choice 2 The antihistamine of choice is oral hydroxyzine hydrochloride initially at 600 mg three times daily, followed by gradual reduction to a minimum maintenance dose required to keep the horse free of lesions.

**DERMATITIS AND DERMATOSIS**

**Etiology**

Some of the identifiable occurrences of dermatitis in food animals and horses are as follows.

**All species**

* Mycotic dermatitis due to *Dermatophilus congolens* , in horses,cattle, sheep
* *Staphylococcus aureus* is a common finding in cases in all species, either as a sole pathogen or combined with other agents
* Ringworm
* Photosensitive dermatitis
* Chemical irritation (contact dermatitis) topically
* Arsenic - systemic poisoning
* Mange mite infestation - sarcoptic, psoroptic, chorioptic, demodectic mange
* Strongyloides (Pelodera) sp. dermatitis

**Cattle**

* Udder impetigo - *S. aureus*
* Cowpox
* Ulcerative mammillitis - udder and teats only
* Lumpy skin disease
* Foot-and-mouth disease – vesicles around natural orifices; vesicular

stomatitis with lesions on teats and coronet.

* Rinderpest, bovine virus diarrhea, bovine malignant catarrh, bluetongue- erosive lesions around natural orifices, eyes, coronets
* Perianal vesicular and necrotic dermatitis associated with mushroom poisoning
* Dermatitis on legs - potato poisoning, topical application of irritants or defatting agents.
* Bovine exfoliative dermatitis.
* Slurry heel.

**Sheep and goats**

* Strawberry foot rot – Dermatophilus
* Sheep pox
* Contagious ecthyma
* Ulcerative dermatosis
* Rinderpest. peste de petits ruminants, bluetongue
* Foot and mouth disease and vesicular stomatitis
* Fleece rot - constant wetting and associated with P aeruginosa
* Lumpy wool ; D. congolensis
* Itch-mite infestation
* Blowfly infestation (cutaneous myiasis)
* Louse infestation.
* Ovine atopic dermatitis
* Caprine idiopathic dermatitis
* Post dipping necrotic dermatitis

**Horses**

* Staphylococcus hyicus in a syndrome reminiscent of greasy heel
* Actinomyces viscosum
* Horse pox
* Viral papular dermatitis
* Vesicular stomatitis - vesicles around natural orifices
* Sporotrichosis
* Dermatophytes, including ringworm.
* Atopic dermatitis (IgE-mediated hypersensitivity)
* Chronic eosinophilic dermatitis
* dermatitis and stomatitis.
* Cutaneous habronemiasis
* Ulcerative dermatitis, thrombocytopenia and neutropenia in

neonatal foals

**Special local dermatitides**

These include dermatitis of the teats and udder, the bovine muzzle and coronet, and flexural seborrhea, and are dealt with under their respective headings.

**CLINICAL FINDINGS**

Affected skin areas first show erythema and increased warmth. The subsequent stages vary according to the type and severity of the causative agent. There may be development of discrete vesicular lesions or diffuse weeping. Edema of the skin and subcutaneous tissues may occur in severe cases. The next stage may be the healing stage of scab formation or, if

the injury is more severe, there may be necrosis or even gangrene of the affected skin area. Spread of infection to subcutaneous tissues may result in a diffuse cellulitis or phlegmonous lesion. A distinctive suppurative lesion is usually classified as pyoderma. Deep lesions which cause damage to dermal collagen may cause focal scarring and idiopathic fibrosing dermatitis.

A systemic reaction is likely to occur when the affected skin area is extensive. Shock, with peripheral circulatory failure, may be present in the early stages. Toxemia, due to absorption of tissue breakdown products, or septicemia due to invasion via unprotected tissues, may occur in the later stages.

**Clinical pathology(Diagnosis)**

Examination of skin scrapings or swabs for parasitic, bacterial or other agents is essential. Culture and sensitivity tests for bacteria are advisable to enable the best treatment to be selected. Skin biopsy may be of value in determining the causal agent. In allergic or parasitic states thereis usually an accumulation of eosinophils in the inflamed area. In mycotic dermatitis organisms are usually detectable in the deep skin layers although they may not be cultivable from superficial specimens

TREATMENT

Primary treatment must be to remove the noxious physical or chemical agent from the environment or to supplement the diet to repair a nutritional deficiency

The choice of a suitable treatment for infectious skin disease will depend upon the accurate identification of the etiological agent

Supportive treatment includes both local and systemic therapy. Local applications may need to be astringent either as powders or lotions in the weeping stage or as greasy salves in the scabby stage. The inclusion of corticosteroids or antihistamine preparation is recommended in allergic states and it is desirable toprescribe sedative or anesthetic agents when pain or itching is severe If shock is present, parenteral fluids should be administered. If the lesions are extensive or secondary bacterial invasion

is likely to occur, parenterally administered antibiotics or antifungal agents may bepreferred to topical applications. To facilitate skin repair, a high protein diet or the administration of protein hydrolysates or amino acid combinations may find a place in the treatment of valuable animals. The use of vaccines as prophylaxis in viral and bacterial dermatitides must not be neglected. Autogenous vaccines maybe most satisfactory in bacterial infections.

An autogenous vaccine is particularly recommended in the treatment of

staphylococcal dermatitis in horses and bovine udder impetigo in which long and repeated courses of treatment with penicillin produce only temporary remission.

An autogenous vaccine produces a cure in many cases.

**Photosensitization**

Photosensitization occurs when skin (especially areas exposed to light and lacking significant protective hair, wool, or pigmentation) becomes more susceptible to ultraviolet light because of the presence of photodynamic agents. Photosensitization differs from sunburn and photodermatitis, because both of these conditions result in pathologic skin changes without the presence of a photodynamic agent.

Photosensitization is typically classified according to the source of the photodynamic agent. These categories include primary (type I) photosensitivity, aberrant endogenous pigment synthesis (type II) photosensitivity, and hepatogenous (secondary, type III) photosensitivity. A fourth category termed idiopathic (type IV) photosensitivity has been described.

A wide range of chemicals, including some that are fungal and bacterial in origin, may act as photosensitizing agents. However, most compounds that are important causes of photosensitivity in veterinary medicine are plant-derived. Photosensitization occurs worldwide and can affect any species but is most commonly seen in cattle, sheep, goats, and horses.

**Primary photosensitization**

Photosensitization due to the ingestion of exogenous photodynamic agents usually occurs when the plant is in the lush green stage and is growing rapidly. Livestock are affected within 4-5 days of going onto pasture and new cases cease soon after the animals are removed. In most cases the plant responsible must be eaten in large amounts and will therefore usually be found to be a dominant inhabitant of the pasture. All species of animals are affected by photodynamic agents, although susceptibility may vary between species and between animals of the same species.

Photosensitizing substances that occur naturally in plants include:

**Photosensitization due to a berrantpigment synthesis**

The only known example in domestic animals is inherited congenital porphyria, in which there is an excessive production in the body of porphyrins, which are photodynamic.

**Hepatogenous photosensitization**

The photosensitizing substance is in all instances phylloerythrin, a normal end product of chlorophyll metabolism excreted in the bile. When biliary secretion is obstructed by hepatitis or biliary duct obstruction, phylloerythrin accumulates in the body and may reach levels in the

skin that make it sensitive to light.

Although hepatogenous photosensitization is more common in animals grazing grep.pasture, it can occur in animals fed entirely on hay or other stored feeds and in animals exposed to hepatotoxic chemicals e.g. carbon tetrachloride. There appears to be sufficient chlorophyll, or breakdown products of it, in stored feed to produce critical tissue levels of phylloerythrin in affected animals.

The following list includes those substances or plants that are common causes of hepatogenous photosensitization.

The individual plants are discussed in more detail in the section on poisonous plants.

* Plants containing hepatotoxins
* Plants containing steroidal saponins
* Congenitally defective hepatic function
* Photosensitization of uncertain etiology

**Clinical finding**

**1-General signs**

These commence with intense irritation and the animal rubs the affected parts, often lacerating the face by rubbing it in bushes. When the teats are affected the cow may kick at them and walk into ponds to immerse the teats in water, sometimes rocking backwards and forwards as if to cool the affected parts. In nursing ewes there may be resentment towards the lambs sucking, and heavy lamb mortalities due to starvation may result.

Local edema is often severe and may cause drooping of the ears, closure of the eyelids and nostrils, causing dyspnea, and dysphagia due to swelling of the lips. An early sign is increased lacrimation, with the initially watery discharge developing into a thicker, serous discharge accompanied by blepharospasm and swelling of the eyelids.Initial erythema of the muzzle is followed by fissuring, then sloughing of the thick skin.

**2-Skin lesions**

Skin lesions are initially erythema, followed by edema and subsequently weeping with matting then shedding of clumps of hair, and finally gangrene. They have a characteristic distribution, restricted to the

unpigmented areas of the skin and to those parts which are exposed to solar rays. They are most pronounced on the dorsum of the body, diminishing in degree down the sides and are absent from the ventral surface. The demarcation between lesions and normal skin is very clear-cut, particularly in animals with broken-colored coats.

Predilection sites for lesions are the ears, conjunctiva, causing opacity of the lateral aspect of the cornea, eyelids, muzzle,face, the lateral aspects of the teats and, to alesser extent, the vulva and perineum. In solid black cattle dermatitis will be seen at the lips of the vulva and on the edges of the eyelids, and on the cornea. Linear erosions often occur on the tip and sides of the tongue in anin1als with unpigmented oral mucosa. In severe cases the exudation and matting of the hair and local edema causes closure of the eyelids and nostrils.In the late stages necrosis or dry gangrene of affected areas leads to sloughing of large areas of skin.

**3-Systemic signs**

These include shock in the early stages,due to extensive tissue damage. There isan increase in the pulse rate with ataxia and weakness. Subsequently a considerable elevation of temperature (41-42°C,106-107°F) may occur.

**4-Nervous signs** These including ataxia, posterior paralysis and blindness; depression or excitement are often observed. A peculiar sensitivity to water is sometimes seen in sheep with facial eczema: when driven through water they may lie down in it and have a convulsion.

**5-Liver insufficiency**

Signs are described elsewhere and may accompany photosensitive dermatitis when it is secondary to liver damage.

**Diagnosis**  There are no suitable field tests to determine whether or not photosensitivity is present.

Hepatogenous photosensitization can be diagnosed by analysis of plasma phylloerythrin concentration using a spectroscopic method. Plasma or serum fluorescence can be used to measure the elevation of phylloerythrin above normal levels prior to hepatogenous photosensitization. Levels in skin are also increased.

**Differential diagnosis**.

• Mycotic dermatitis

• Frequent wetting, as in periods of heavy rainfall, along the back in horses or cattle with a dense hair coat

• Bighead of rams associated with Clostridium novyi infection

• The eye lesions in photosensitization have been confused with those of pinkeye but that disease is not accompanied by extensive dermatitis.

**Treatment**

Primary treatment includes immediate removal from direct sunlight, prevention of ingestion of further toxic material and the administration of laxatives to eliminate toxic materials already eaten. In areas where the disease is enzootic the use of dark-skinned breeds may make it possible to utilize pastures that would otherwise be too dangerous.

Local treatment will be governed by the stage of the lesions. Nonsteroidal anti- inflammatory drugs (NSAIDs), corticosteroids or antihistamines can be administered parenterally and adequate doses maintained. To avoid septicemia the prophylactic administration of antibiotics may be worthwhile in some instances.

**Alopecia and hypotrichosis**

**Etiology**

Alopecia is complete absence of the hair or wool coat; hypotrichosis is less than the normal amount of hair or wool. Both may be caused by the following conditions.

1- **Failur of follicles to develop**

- Congenital hypotrichosis.

**2-Loss of follicles**

-Cicatricial alopecia due to scarring after deep skin wounds that destroy follicles. Cicatricial alopecia occurs following permanent destruction of the hair follicles, and reg rowth of hair will not occur. Examples include physical, chemical or thermal injury, severe furunculosis, neoplasia and certain infections such as cutaneous onchocerciasis.

**3- Failure of the follicle to produce a fiber**

* Inherited symmetrical alopecia
* Congenital hypotrichosis
* Hair- coat-color-linked follicle dysplasia
* Inherited dyserythropoiesis and dyskeratosis
* In baldy calves and adenohypophyseal hypoplasia
* Congenital hypothyroidism (goiter) due to iodine deficiency in the dam.
* After viral infection of the dam, alopecia congenitally in the newborn, e.g. after bovine virus diarrhea in cattle and infection by a similar virus in sheep (Border disease).
* Neurogenic alopecia due to peripheral nerve damage
* Infection in the follicle

**4- Loss of preformed fibers**

* Dermatomycoses – ringworm

Mycotic dermatitis in all species due to D. congolensis

* Metabolic alopecia subsequent to a period of malnutrition
* Idiopathic hair loss from the tail-switch of well- fed beef bulls In sterile eosinophilic folliculitis of cattle
* Wool slip

**Clinical findings**

When alopecia is due to breakage of the fiber, the stumps of old fibers or developing new ones may be seen. When fibers fail to grow the skin is shiny and in most cases is thinner than normal.

In cases of congenital follicular aplasia, the ordinary covering hairs are absent but the coarser tactile hairs about the eyes, lips and extremities are often present. Absence of the hair coat makes the animal more susceptible to sudden changes of environmental temperature.

There may be manifestations of a primary disease and evidence of scratching or rubbing. Congenital hypotrichosis results in alopecia which is apparent at birth or develops within the neonatal period.

**CLIN ICAL PATHOLOGY(Diagnosis)**

If the cause of the alopecia is not apparent after the examination of skin scrapings or swabs, a skin biopsy will reveal the status of the follicular epithelium.

Diagnostic confirmation of alopecia is by visual recognition, the diagnostic problem being to determine the primary cause of the hair or

fiber loss. Hypotrichosis is a reduction in numbers of fibers instead of a complete absence.

**TREATMENT**

Primary treatment consists of removing the causes of trauma or other damage to fibers. In cases of faulty follicle or fiber development treatment is not usually attempted.