Culicoides Hypersensitivity in the Horse: 15 Cases in Southwestern British Columbia

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Summary

The investigation of a chronic, seasonal dermatitis of horses in southwestern British Columbia is described. Typically the history indicated an insidious onset, followed by a gradual progression in the severity of the signs each year. Lesions appeared during the warmer months of the year and tended to regress during the winter. The clinical signs consisted of areas of pruritus and excoriation, affecting predominantly the ventral midline, mane and tailhead. In all cases corticosteroid therapy relieved the pruritus and allowed the lesions to heal.

The salient pathological findings were hyperkeratosis, spongiosis and a dermal infiltration of eosinophils together with mononuclear cells. These changes are typical of an allergic dermatitis, which has been recognized in many parts of the world as a hypersensitivity reaction to the bites of *Culicoides* spp. In this instance, the epidemiological findings relating to the geographic area, the local insect population and the distribution of lesions implicated *Culicoides obsoletus* as the etiological agent.

Key words: Horses, allergic dermatitis, *Culicoides obsoletus*, hypersensitivity, *Ceratopogonidae*.

Résumé

Rapport de 15 cas d'hypersensibilité à l'endroit de *Culicoides*, chez des chevaux du sud-ouest de la Colombie Britannique

Cet article décrit l'investigation relative à une dermatite chronique et saisonnière qui affectait des chevaux du sud-ouest de la Colombie Britannique.

L'anamnèse mentionnait infailliblement un début insidieux, suivi par une aggravation progressive des signes cliniques, à chaque année. Les lésions apparaissaient durant les mois les plus chauds et s'estompaient graduellement au cours de l'hiver. Les signes cliniques se traduisaient par des zones de prurit et d'excoriation, localisées surtout sur la ligne blanche abdominale, dans la crinière et à la base de la queue. Dans tous les cas, l'utilisation de corticostéroïdes élimina le prurit et les lésions.

L'examen microscopique de biopsies cutanées révéla la présence d'hyperkératose et de spongiosité, ainsi qu'une infiltration du derme par des éosinophiles et des mononucléaires. Ces lésions sont caractéristiques d'une dermatite allergique, assimilée dans plusieurs parties du monde à une réaction d'hypersensibilité aux morsures d'insectes du genre *Culicoides*. Dans le cas présent, les constatations épizootiologiques relatives à la région géographique, la population locale d'insectes et la localisation des lésions incriminaient *Culicoides obsoletus* comme agent étiologique.

Mots clés: chevaux, dermatite allergique, *Culicoides obsoletus*, hypersensibilité, *Ceratopogonidae*.

Introduction

Seasonal dermatitis in horses was first observed in the East Indies by Kerr in 1860 [1] and since that time it has been reported throughout the world [1-10]. A variety of names and suspected etiologies have been ascribed to the condition, but in all instances the clinical signs were similar, differing only in the location of lesions (Table I). It can be seen from Table I that the mane and tail were almost always affected, while lesions on the ventral midline and other parts of the body were less commonly reported. Observations on the epidemiology of the disease show that it occurs in temperate climates during the warmer months of the year [1-16]. It is usually seen in horses that are kept outside and tends to affect only one or two horses in a group. A genetic predisposition has been noted [4,9,17,18].

The first commonly proposed etiology of equine recurrent dermatitis was *Onchocerca cervicalis* [3,5,8]. The possible significance *O. cervicalis* microfilariae in skin biopsies of affected areas has remained controversial. However, Riek [19] proved conclusively, as others have since [12-16], that the causative agent was *Culicoides* spp. Other insects such as *Stomoxys calcitrans* and *Haemotobia irritans* may also be involved [16,20].

The present study is a report of 15 horses in southwestern British Columbia with *Culicoides* hypersensitivity (CH) and a comparison of the syndrome in western Canada with that seen in other parts of the world.

History

Twelve horses were presented because of a chronic pruritic dermatitis. Three horses were presented for other reasons. In most instances the condition had an insidious onset and the exact duration of the problem was often not known. All the affected horses had signs during the summer months. Many displayed signs beginning in spring and lasting through fall; three had signs all year. In six horses

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the signs became progressively more severe and extended for a longer period each year. Two horses had deteriorated markedly over a three year period. The most severe lesions varied in location from year to year but the area observed during the clinical examination was usually the initial site of clinical lesions. No breed or sex predilection was observed. Thirteen horses were pastured with unaffected horses.

Clinical Signs

The area of skin commonly affected was the ventral midline (thirteen), followed by the tailhead (eight), the mane and withers (six), the head and ears (three), shoulders (two) and over the tuber coxae and chest (one). Two involved the ventral midline only, whereas in eight animals the ventral midline was affected in combination with the mane and/or the tail. On the mane, lesions were usually confined to the caudal portion and adjacent withers. When the tailhead was affected, it often involved an area 20 cm by 20 cm and extended onto the proximal third of the tail.

Regardless of the site on the body, all the lesions had similar appearance and were characterized by intense pruritus with localized alopecia and evidence of excoriation (Figures 1 and 2). In eight horses, self inflicted trauma was associated with serous effusion and the formation of crusts (Figure 2). The areas of alopecia were covered by hair stubbles of varying lengths. Lichenification was a very common finding, especially on the ventral midline (Figure 2). Seborrhea sicca and a dry, rough hair-coat was noted in three horses.

Laboratory Analysis

The protocal for all 15 horses was similar. A punch biopsy of skin 8 mm in diameter and a skin scraping were taken from each of the affected areas. Biopsy specimens were divided into two pieces. One was preserved in 10% buffered formalin and submitted for histopathology.[1,2] The other piece was minced with a scalpel and incubated with 0.5 mL of saline at room temperature. Two to six hours later, the saline was examined microscopically for the presence of microfilariae. The samples from horses seven and eight contained microfilariae.

Material from the skin scrapings was examined for the presence of ectoparasites and pathogenic fungi; none was observed.

Transparent adhesive tape was applied to the perineal area and examined for *Exyuris equi* eggs, but none was seen.

A blood sample for a complete blood count (CBC) was taken from each of four horses. An eosinophilia was consistently observed.

Histopathology

The samples of epidermis of all the horses had hyperkeratosis, usually associated with spongiosis, but rarely with parakeratosis (Figures 3 and 4). Several specimens had local necrosis of the epidermis. Evidence of excoriation was seen frequently, but crusts

TABLE 1
SUMMARY OF THE IMPORTANT LITERATURE ON SEASONAL DERMATITIS IN THE HORSE

Year	Country	Author	Name of Seasonal Dermatitis	Proposed Etiology	Area Affected (in order of frequency)
1891	Australia	Bancroft	Queensland itch	biting insects	mane, tail
1924	United States	Freeborn *et al*	Bursatae	hereditary, not nematodes	forelegs, hindlesgs, trunk
1934	Philipines	Underwood	Dhobie itch	microfilariae in skin	poll, mane, neck, breast, shoulders, withers, ventral midline, thighs, head, croup, back
1937	France	Henry *et al*	Summer sores	hereditary	midline, from poll to tail, rarely ventral midline
1939	India	Datta	Lichen tropicus	*O. cervicalis* microfilariae	
1946	France	Vails			
1947	Algeria	Le Seach	Summer dermatitis	unknown, not hereditary	anterior cannon bone
1948	United States	Dikmans	Summer dermatitis	*O. cervicalis*	neck, breast, ventral midline, legs
1953	Australia	Riek	Queensland itch	*culicoides robertsi*	mane, tail, poll
1954	Israel	Ralbag	Allergic uritcaria	*Mosquitoes* spp	mane, tail, poll
1956	Japan	Nakamura, Matsuhashi *et al*	Kasen	*Culicoides* spp	neck, withers, tail
1964	Germany	Becker	Summer eczema	possibly small flying insects	mane, withers, tail, back, neck, shoulders, chest, ventral midline, head
1971	United States	McMullan	Allergic dermatitis	*Culicoides,* spp	mane, tail, poll
1974	England	Mellor, McCaig	Sweet itch	*Culicoides pulicaris*	mane, tail
1977	United States	Stannard, Baker	Culicoides hyper-sensitivity	*Culicoides* spp	mane, tail, poll
1978	Ireland	Baker, Quinn	Sweet itch	*Culicoides* spp	mane, tail, poll, lateral aspects of body

[1]Dr. A.A. Stannard, Dermatopathology Service, Davis, California.
[2]British Columbia Veterinary Laboratory, Abbotsford, British Columbia.

with dermal hyperplasia were seen in only three cases.

Eight horses had a dermal infiltration of eosinophils, sometimes in combination with macrophages (Figure 5). Excoriation was accompanied by greater numbers of dermal inflammatory cells. In case two there was proliferation of small blood vessels in the dermis, and some evidence of arteritis.

FIGURE 1. Ventral midline of horse 15; lichenification, alopecia, excoriation and crusts.

FIGURE 2. Closer view of the horse in Figure 1.

Diagnosis

The diagnosis of *C. obsoletus* hypersensitivity was made and is supported by the following findings. First and foremost, the epidemiology, history and clinical signs are similar to those reported elsewhere (4,8,9,11-16). Secondly, *C. obsoletus* is very common in southwestern British Columbia and is virtually the only species of *Culicoides* present in this mild temperate area (21). Furthermore, *C. obsoletus* has been reported to be the most common species of *Culicoides* found on the horse (14). This species prefers to feed on the ventral midline, though a few will sometimes feed on the withers and haunches (14), which corresponds with the distribution of the lesions observed.

No pathogens were found in skin scrapings or in transparent tape examinations. In only two of the 15 horses were microfilariae seen in incubated minced biopsy specimens, or on histopathology. Therefore, it is unlikely that *Onchocerca* spp was of prime importance in the etiology. On laboratory examination, the most common diagnosis made by the dermatopathologist was "allergic dermatitis, probably due to *Culicoides*", whereas the general pathologist's diagnosis was usually "dermatitis".

The data presented are consistent with a hypersensitivity to insect bites, probably *C. obsoletus*. Other insects which must be considered are *Haemotobia irritans* (horn fly) and *Stomoxys calcitrans* (stable fly). The horn fly has been reported to cause an allergic dermatitis but only of the ventral midline and neck (15,22). It is therefore unlikely that it was the sole agent involved, but it is possible that it contributed to the clinical signs observed. The stable fly causes a very severe pruritus, with lesions on the back, chest, neck and legs (22). Significant intradermal reactions to *Stomoxys calcitrans* extract have been observed in cases of equine recurrent dermatitis (16,20,23).

Treatment and Outcome

Oral prednisolone[3] was prescribed at 400 mg per animal two times per day for five days. A corticosteriod cream prepared from an ointment base[4] and 9-fluoroprednisolone acetate[5] (0.1 mg/g) was applied to the affected areas two times per day until the signs resolved. Clients were instructed to gradually decrease the dose of oral prednisolone after the initial five day treatment period to determine the lowest dosage rate that would prevent pruritis. The immediate

[3]Deltazone, Upjohn Company, Orangeville, Ontario.
[4]Dermabase, Bordon Company, Don Mills, Ontario.
[5]Pre-def 2-X, Upjohn Company, Orangeville, Ontario.

response to therapy was good. The pruritus was usually absent two days after therapy was initiated with the hair coat returning to normal within a month. Most horses were maintained on a total dose of 400-500 mg or oral prednisolone on alternate days. Only case 10 failed to respond, therefore, in this animal 10 mg of dexamethazone[6] was administered two times per day for four days, followed by 800 mg of prednisolone *per os* on alternate days. A good response was observed.

Prevention

Prophylactic measures to reduce the exposure to *Colicoides* are preferable to long-term corticosteroid therapy. During the fly season, clients were encouraged to keep their horses stabled from three hours before dusk to two hours after dusk. This is when most *Culicoides* spp feed, though some feed throughout the day (24). *Culicoides obsoletus* is particularly active at dusk (14), so it is logical to stable horses at this time of day. In six horses in which this minimal prevention program was used there was a marked reduction in the severity of clinical signs within one to two weeks. Ideally, the stable doors and windows should be covered with a very fine screen; ordinary mosquito screen is not effective (15, Dr. A.A. Stannard, personal communication). However, *Culicoides* spp are uncommon indoors (18) and stables without screens have been used successfully in treatment (9,14,16,24,25) with most reports indicating that stabled horses are not affected (9,12,14,16,25,26). The preventive measures required to treat or prevent CH depend both on the severity of the lesions and the number and species of *Culicoides* present.

Many clients had no stables and preferred to use topical fly repellents every afternoon or to attach fly repellent tags to the mane, tail or ventral midline. Both measures were inconsistent in the prevention of CH, the former because of poor client compliance, and the latter because the tags were ineffective in repelling *Culicoides* spp. Consequently, it was necessary for many horses to be maintained on prednisolone therapy.

Piperonyl butoxide spray used twice daily (27) and DDT spray used weekly (28) have each been reported to be effective in preventing CH.

FIGURE 3. Hyperkeratosis, parakeratosis, spongiosis and ballooning degeneration are very prominent. X530.

Discussion

The epidemiology noted was typical of previous reports of CH (1-5,7-16). Ponies did not seem to be more susceptible, which is inconsistent with what has been reported in England and Ireland (16,29). Only case 12 was a pony. The horses in the study were not related. A familial predisposition, correlated more with the dam than with the sire, has been observed (4,9,15,17,18). Most reports indicate that there is no sex predilection (18,30). Dark coat color has been noted to be significant by some (11,12) but not by others (9,30). In our study, 12 of the 15 cases were bays or chestnuts. Objective data on the color distribution of the general clinic caseload does not exist; therefore, a valid comparison cannot be made. Older horses were the most severely affected, which is consistent with earlier reports on
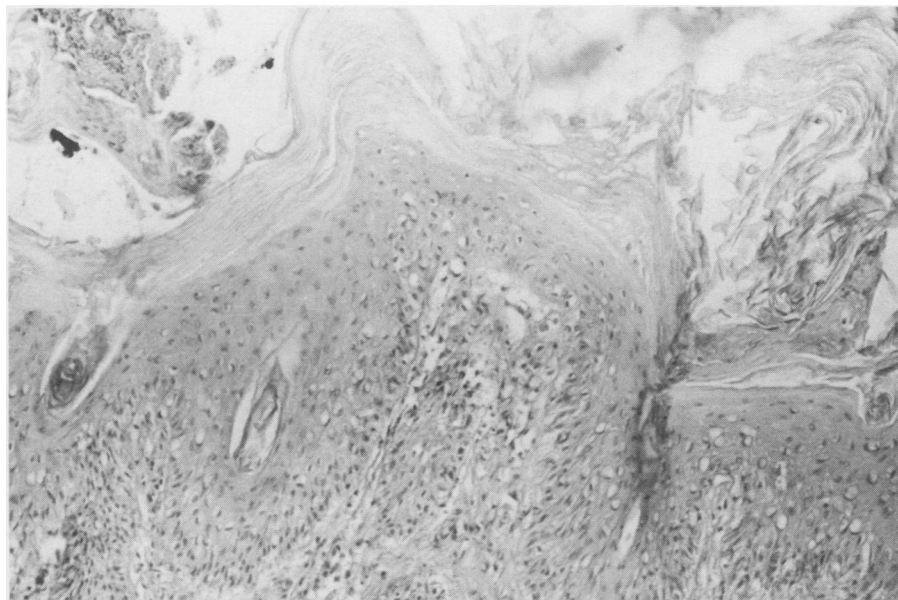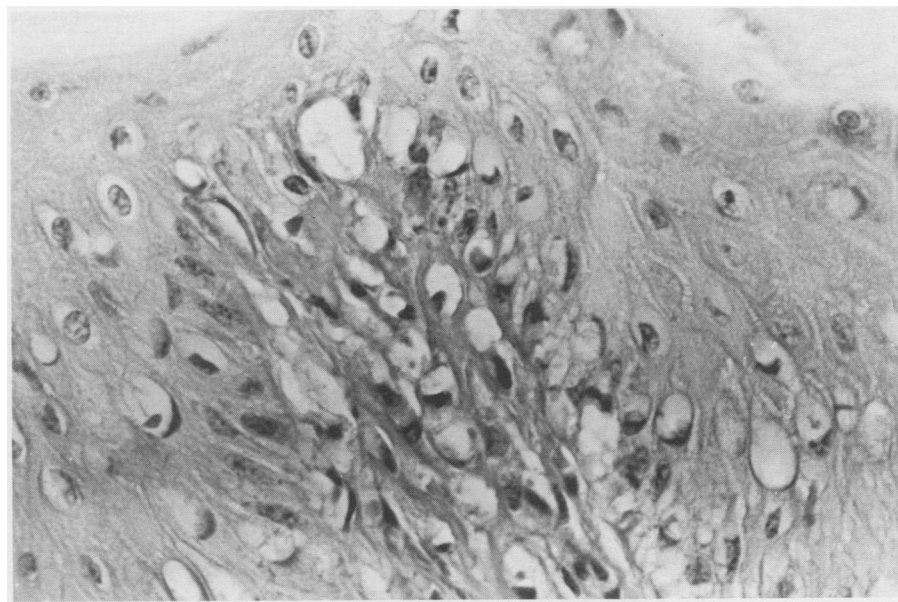

FIGURE 4. Higher magnification of Figure 3; spongiosis and ballooning degeneration. X1330.

[6]Azium, Schering Canada, Pointe Claire, Quebec.

TABLE II
EPIDEMIOLOGY, HISTORY AND CLINICAL SIGNS OF AFFECTED HORSES

Case	Age	Color	Horses Affected/ No. on Farm	Management	Known Duration of Problem	Season Affected	Time Biopsy Taken	Area Involved and Clinical Signs	Comments
1	11	Chest	1/4	Outside except at night	3 yrs.	Spring Summer Fall	April	VM^{a}a^b,e^c,l^d,c^e Tailhead a,e,l Ears a,e Shoulders	clinical signs rapidly worsened each year
2	6	Roan	2/12	Always outside	> 10 mo.	All year	July	V.M. a,e,l,c	
3	14	Chest	1/2	Outside except at night	4 yrs.	Spring Summer Fall	Oct.	V.M. a,e,l Tailhead a,e,l Seborrhea sicca Rough haircoat	
4	14	Bay	1/8	Outside except at night	> 2 yrs.	Summer Early Fall	Oct.	V.M. a,e,l,c Tailhead a,e,l Mane a,e Chest a,e Shoulders a Seborrhea sicca	horse broke large window and cut himself while rubbing tail
5	5	Bay	1/2	Always Outside	2 yrs.	Spring Summer Fall	Oct.	V.M. a,e	
6	7	Bay	2/35	Outside during summer	Not known	Summer	July	V.M. a,e Tailhead a Dry skin	
7	3	Bay	1/6	Always outside	2 wks.	Summer	July	V.M. a,e	mainly microfilariae seen
8	18	Chest	1/1	Always outside	> 8 yrs.	All year	May	V.M. a,e,l,c Hyperpigmentation Forehead ^{a}	hyperpigmentation prominant on V.M. 4 microfilariae seen on minced sample from V.M. but no microfilariae seen on any histological sections
9	9	Bay	1/2	Outside except at night	4 mos.	Summer	Nov.	V.M. a	
10	6	Bay	1/2	Outside except at night	18 mos.	All year	July	V.M. a,e,l,c Neck (nodules)	No response to prednisolone therapy; required dexamethazone to decrease pruritus. Nodular necrobiosis on neck.
11	9	Bay	1/2	Always outside	2 yr.	Spring Summer	June	Mane a,e Body — small nodules	Nodular necrobiosis all over body
12	13	Grey	1/?	Always outside	3 yr.	Spring Summer Fall	June	V.M. a,e,l,c Erosions Tailhead a,e,l,c Withers a,e,l,c Tuber coxae a,e Head a,e	Clinical signs rapidly worsened each year
13	4	Grey	2/35	Always outside	1 yr.	Summer	Sept.	V.M. a,l Tailhead a,e Mane a,e	Lack of eosinophils seen on histology
14	3	Chest	1/2	Outside except at night	2 yr.	Summer Fall	Oct.	Mane a,e Tailhead a,e,c	
15	5	Chest	1/4	Always outside	2 yr.	Summer	Aug.	V.M. a,e,l,c Mane a,e Tailhead a,e,c	Ulceration very prominant on histopathology

^{a} ventral midline
^{b} alopecia
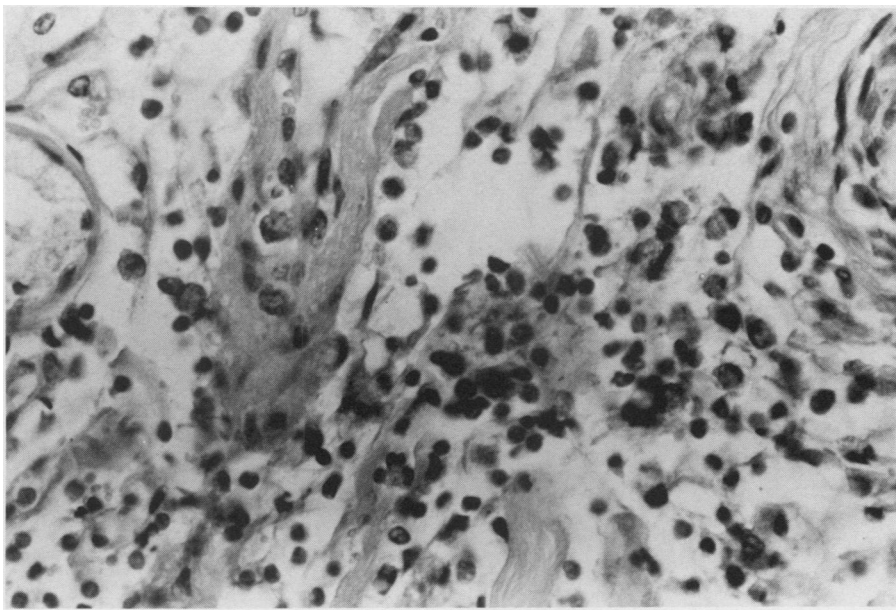^{c} excoriation
^{d} lichenification
^{e} crusts

FIGURE 5. Dermal infiltration of eosinophils. X1330.

Culicoides hypersensitivity (4,9,15,16) and hypersensitivity reactions in general (31). Three horses had signs throughout the year which has been reported in very severe cases (15,30).

The clinical signs observed differed from previous reports only in the predominance of lesions on the ventral midline. Ventral midline lesions have been reported to occur less frequently than those on the mane and tail (3,4,5,8) or not to exist at all (2,9-11,13-16). Only cases that were biopsied were included in this study. Due to the problems in biopsying the mane and tail, relatively few were done. This study has therefore overemphasized the lesions on the ventral midline.

The histology was typical of the type I hypersensitivity which is commonly seen in CH (16). Although an eosinophilia was observed in this study, and increased serum concentrations of histamine have been noted previously, treatment with antihistamines has generally been unsuccessful (32,33).

Four microfilariae were seen on histology of the specimen, from horse 9. This supports a diagnosis of onchocerciasis; there were no seasonal variations in clinical signs. A diagnosis of CH is supported by the relatively low numbers of microfilariae as compared to confirmed cases of onchocerciasis, and even clinically normal horses (16,19,34). Stannard and Cello have found that within a geographical region horses having significantly higher than average numbers of microfilariae had lesions associated with the infection (35). In a survey of over 300 horses in western Canada, 12% had microfilariae (as determined by ventral midline skin punch biopsies), while clinical signs were rarely observed, (Polley, L., personal communication). Furthermore, the severe pruritus, alopecia, excoriation, lichenification, crusts and large area involved are more typical of CH (15,34). It is possible that the two syndromes coexist in case 8.

Many microfilariae were seen on histology of the specimen from horse 7, supporting a diagnosis of onchocerciasis. However, the sudden onset of clinical signs and the presence of lesions only during the summer suggests CH (15,22,34). Both case 7 and 8 responded well to oral corticosteroid therapy and were free of lesions throughout the winter which is more consistent with CH.

Several questions on CH remain unanswered. Why is *C. obsoletus* less pathogenic in England than in southwestern British Columbia? It is very hard to identify the subspecies of *C. obsoletus* and this was not done in either of the studies referred to earlier, in England and British Columbia (14,21). It is possible that the subspecies present in British Columbia is different from the one(s) in England. To answer this problem would require intradermal skin testing, comparing extracts prepared from different subspecies.

In order to make a definitive diagnosis of most skin allergies it is necessary to do active tests for skin hypersensitivity in both normal and affected horses (16). This is done using intradermal injections of insect extracts. There is no commercial source of *Culicoides* spp, *Stomoxys calcitrans* or *Haemotobia irritans* extract, but a simple procedure for preparing one has been described (16,22). The results from this stage of our investigation are incomplete. Intradermal skin testing is impractical since it is usually possible to diagnose CH without it.

More specific data are required on the minimal treatment and preventive measures needed. The mode of genetic transmission must also be discerned. The greatest challenge lies in the hyperimmunization or desensitization of effected horses. Much work has been done in this area, but with no success (16,22).

Culicoides hypersensitivity has been frequently seen by the authors in southwestern British Columbia. The 15 horses reported were the worst that were observed in a three year period; over 125 other horses were affected to a lesser degree. The scant attention given to the disease in Canada may give the impression that the disease has little significance but severely affected horses often cannot be worked as the pruritus may be exacerbated by sweating (9). Certain horses cannot wear a saddle due to lesions on the withers and ventral midline. Furthermore, pruritic horses often develop dangerous vices in an attempt to scratch affected areas (9). The cost of treatment and prevention is also very significant. An average alternate-day dose of prednisolone (500 mg) is worth $1.57 (wholesale cost). The inconvenience of keeping a horse stabled when pasture is available is greater yet. The greatest cost arises from the damage that pruritic horses will do to such things as fences and gates and to themselves.

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