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An analysis of factors underlying hypotrichosis and alopecia in Irish Water Spaniels in the United Kingdom

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Abstract A survey on the occurrence of dermatoses in the Irish Water spaniel (IWS) was carried out in the United Kingdom. A group of 20 dogs was selected and examined clinically. All dogs had a nonpruritic, noninflammatory, regionalized hair loss affecting the same areas of the body in males and females, although an initial cyclical pattern associated with the oestrus cycle was identified. Hormonal investigations showed features suggestive of an abnormality of steroidogenesis. Histopathology revealed features similar to canine recurrent flank alopecia (CRFA) and follicular dysplasia associated with abnormal melanization, as in colour dilution alopecia, although the clinical features did not correlate with those conditions. Dietary changes improved coat and skin quality in most of the cases in this series but the role of the diet was not investigated further. This study suggests that hair loss in IWS is influenced by dietary factors and sexual hormones. Abnormalities of the steroidogenic pathways may contribute to the severity of the condition.

Keywords: alopecia, skin, dog.

INTRODUCTION

The Irish Water Spaniel (IWS) is a breed that has been present in Ireland since the last century. The first description of a water spaniel is in the 'De Canibus Britannicis' (1570), although the progenitor of the breed is a dog which was born in 1834 in Dublin.[1] The British Kennel Club Breed Standard for IWSs was first published in 1870 and emphasis was placed on the presence of a bare tail as a characteristic for this breed. The standard was subsequently modified in 1950 to include the presence of a smooth throat. Nowadays the British Breed Standard states: 'the throat should be smooth and the smooth hair forming a V-shaped patch from back of lower jaw to breast bone', and the tail should have 'three to four inches at tail root covered by close curls which stop abruptly. The remainder bare or covered by short straight fine hairs'. The coat colour should be dark liver or puce-liver.[2] Similar requisites are detailed in the Irish and American Breed Standards and are considered as special characteristics for this breed. For many years breeders have been selecting animals on the basis of these characteristics but in the last 15 years breeders in the UK have started to recognize coat disorders. Areas of alopecia or hypotrichosis, affecting not only the ventral neck and distal tail, but also the latero-dorsal neck, flanks, dorsum, rump or the caudal part of the thighs, in both males and females have been noted.[3]

The purpose of this study was to describe in detail clinical aspects of hair loss in IWS and to determine underlying factors in this syndrome. Pedigree data relating to affected dogs were also collected and analysed; these data will be reported elsewhere.

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Definition of terms used in the text:
- alopecia: absence of the hair from the skin areas where it is normally present.
- baldness: loss of hair, alopecia; especially absence of hair from the human scalp.
- hypotrichosis: presence of less than the normal amount of hair.

MATERIALS AND METHODS

Epidemiological survey

In 1995 contacts were established between the British IWS Association (IWSA) and the dermatology unit of the Royal Veterinary College (RVC). A questionnaire was sent to all 216 members of the IWSA to

identify animals with skin or coat disorders. Owners were asked to answer questions on skin and coat condition, describing any clinical lesions, their localization, age of onset and duration, aspects of management (diet, grooming and bathing), previous therapy and clinical investigations. From the responses obtained it was possible to divide the dogs into three categories: normal (without any cutaneous problem); coat problems (if affected by nonpruritic hair loss and without any other concurrent cutaneous disease); and skin problems (if affected by any other skin disorder). In both sexes, a smooth throat (Fig. 1) and a bare tail (Fig. 2) were considered 'normal' according to the standard of the breed.

The respondents reporting dogs with a coat problem, were invited to participate in a clinical study.

Clinical study

Animals with coat disorders. In all cases a general and a dermatological examination were performed. Routine dermatological investigations (skin scrapings, hair pluckings, coat brushings, and trichogram) were undertaken in all cases. The Mackenzie brush technique for fungal culture was carried out and Dermasel agar medium (Oxoid CM539) was used. Hair samples were collected for electron microscopy in one case. Briefly, hairs were mounted on stubs, coated with gold and examined in a Hitachi S450 scanning electron microscope. Routine bacteriological culture and antibiotic sensitivity testing as described by Lloyd *et al.*[4] were carried out only when a concurrent pyoderma was present.

Blood investigations. Blood was collected by jugular venepuncture for routine haematological and biochemical investigations. The serum was stored at–20°C prior to hormonal assay (total thyroxine (T4) and thyroid stimulating hormone (TSH), oestradiol, progesterone, and testosterone). An adrenocorticotrophic hormone (ACTH) stimulation test was also carried out in all cases. Blood was collected before and one hour after intravenous administration of 0.25 mg dog^{-1} of tetracosactrin acetate (Synacthen; Ciba) for cortisol and 17-hydroxyprogesterone (17-OHP) determination. Serum samples were sent for standard radioimmunoassay to an external laboratory (SCL Bioscience Services). A TSH stimulation test was subsequently carried out only in cases where the results of the previous analysis were suggestive of hypothyroidism (low basal T4 and/or hypercholesterolemia) (Table 1). Blood was collected before and six hours after intravenous administration of 0.1 UI kg^{-1} bovine TSH (Sigma). Endogenous ACTH was determined only in cases where the concentration of

Figure 1. Normal Irish Water Spaniel showing lack of hairs on the ventral surface of the neck.

Figure 2. Tail of a normal Irish Water Spaniel illustrating lack of hair.

Table 1. Blood cholesterol and hormone levels in IWS with coat disorders

Case ~	Sex	Cholesterol	Total T4 #	TSH #	TSH test Pre	TSH test Post	ACTH (Cortisol)	ACTH test Pre	ACTH test Post	ACTH test (17-OHP) Pre	ACTH test (17-OHP) Post	Oestradiol	Progesterone	Testosterone
1	F	**10.0**†	39	< 0.10		ND	ND	97	176	2.3	**6.3**	42	1.6	0.40
2	F	7.5	26	< 0.10		ND	ND	105	271	< 1.0	**4.8**	< 10	1.2	0.42
3	M	6.7	29	< 0.10		ND	ND	146	225	2.6	**6.9**	**14.6**	1.0	6.4
4	F	**9.5**	37	0.14		ND	ND	164	304	**5.4**	**10.3**	10.0	2.3	0.40
5	NM	5.2	–	–	22	48	**152**	56	265	< 1.0	**6.7**	< 10	1.3	0.66
6	NF	**10.8**	50	0.24	37	53	ND	186	276	2.9	**9.1**	< 10	1.5	0.38
7	F	**10.5**	**55**	**0.48**	21	57	ND	150	345	1.3	**8.4**	< 10	1.1	0.32
8	M	6.5	**12.3**	0.24	**8.8**	**9.7**	**100**	67	189	1.9	**10.4**	< 10	< 1.0	9.6
9	NF	5.1	**11.5**	**0.57**		ND	ND	266	348	1.9	**5.0**	< 10	1.7	0.52
10	NF	5.8	**11.5**	0.10	**19**	**21**	55	168	294	1.3	**8.6**	< 10	1.5	0.52
11	F	7.4	32	0.17		ND	55	71	265	1.2	**8.1**	14.1	< 1.0	0.48
12	F	4.3	22	0.40		ND	ND	161	254	1.8	**9.0**	< 10	< 1.0	0.16
13	NF	5.5	27	0.18		ND	ND	105	246	< 1.0	**5.0**	< 10	< 1.0	0.10
14	NF	6.6	24	0.19		ND	ND	100	322	< 1.0	**5.0**	11.4	< 1.0	0.56
15	M	**9.0**	17.1	0.32	13	40	68	98	282	1.5	**8.6**	< 10	< 1.0	6.4
16	NM	4.5	**7.9**	< 0.10	24	34	ND	108	329	< 1.0	3.9	**14.1**	**3.8**	1.4
17	NF	6.4	40	0.38		ND	ND	104	313	< 1.0	**5.0**	< 10	1.1	0.56
18	M	5.5	–	–	24	49	ND	58	256	< 1.0	**9.8**	< 10	< 1.0	1.6
19	M	**7.6**	–	–	28	39	49	157	302	2.0	**9.8**	< 10	1.7	8.3
20	F	6.1	–	–	27	68	56	47	316	< 1.0	**7.0**	< 10	< 1.0	0.18

~ Dogs no. 9 and 18 are dead.
#In these two columns (–) indicates that a TSH stimulation test was carried out as an initial test.
†Abnormal values are in bold
ND Not done.
Reference range:
Cholesterol: 2.5–7.5 mmol L^{-1}
Total T4: 13–52 nmol L^{-1}
TSH: < 0.41 ng mL^{-1}
Total T4 (post TSH stimulation): increase at least 1.2 times
ACTH: 20–80 pg mL^{-1}
Cortisol (basal): 20–250 nmol L^{-1}
Cortisol (post ACTH stimulation): < 660 nmol L^{-1}
Oestradiol:
Entire or neutered male: < 10 pmol L^{-1}
Female: (anaoestrus): < 10 pmol L^{-1}
(possible follicular activity): > 30 pmol L^{-1}
Progesterone:
Entire or neutered male: < 3 nmol L^{-1}
Female: various with cycle
17-hydroxyprogesterone
(post-ACTH stimulation): < 4.0 nmol L^{-1}; borderline: 4.0–6.0 nmol L^{-1}
Testosterone: Male: 1.5–26 nmol L^{-1}
Neutered male: < 0.5 nmol L^{-1}
Female: < 0.3–0.5 nmol L^{-1}

17-OHP was above the normal value (Table 1). Lower limits of sensitivity for hormonal assays were: cortisol 20 nmol L^{-1}, progesterone 1.0 nmol L^{-1}, 17-OHP 1.0 nmol L^{-1}, testosterone 0.05 nmol L^{-1}, oestradiol 10.0 pmol L^{-1}, total T4 4.0 nmol L^{-1}, ACTH 5.0 and TSH 0.10 ng mL^{-1}. The interassay variation for each assay was: cortisol 3–7 per cent, progesterone 5–15 per cent, 17-OHP 3–11 per cent, testosterone 7–15 per cent, oestradiol 9.4–17.8 per cent, total T4 3.1–5.7 per cent, ACTH 6.1–7.1% and TSH 6.8–14.4%. TSH was determined using a homologous kit, while all other hormones were determined using human kits which have been validated for the dog. Normal reference range for the laboratory is given in Table 1.

Histopathology. Six-millimetre punch biopsy specimens of skin were collected under local anaesthesia using 2% lignocaine (Lignavet, C-Vet). Specimens

Table 2. Analysis of the Wafcol (Fish & Corn) diet

Label information	
Proteins	21%
Ash	7%
Oil	8%
Fibre	5%
Fatty acid analysis*	
Linoleic acid	3.50
Gamma linolenic acid	< 0.01
Dihomogammalinolenic acid	0.05
Arachidonic acid	0.05
Alpha linolenic acid	0.79
Eicosapentanoic acid	0.24
Docosahexaenoic acid	0.30
ω_6/ω_3 ratio	2.71/1

*Assay performed by International Laboratory Services Ltd, UK, on samples of the diet supplied by Wafcol Ltd.

were obtained from one or more alopecic or hypotrichotic areas on the lateral aspects of the neck, rump and thigh. One specimen was collected in each case from a clinically normal area which was most often the lateral thorax (Table 3). Specimens were fixed in 10% formal saline and transferred to one of the authors (PEM) for processing and examination. Seventy-four formalin-fixed biopsy specimens from 19 IWSs were evaluated. These were bisected vertically, along the line of the hair where possible, and wax sections stained with haematoxylin and eosin were prepared from one half of each sample

Table 3. Correlation of follicle activity and morphology with the clinical appearance of hair coat at various skin sites (histology for case 5 was carried out in another laboratory but reviewed by PEM)

Case	Neck	Thorax	Dorsum	Rump	Thigh
1	*alopecic* mixed abnormal	*normal* active n.a.d.	*hypotrichotic* mixed dysplastic	NS	*alopecic* mixed dysplastic
2	*hypotrichotic* mixed dysplastic	*normal* active suspect	*normal* active abnormal	NS	*hypotrichotic* mixed abnormal
3	*hypotrichotic* mixed n.a.d.	*normal* mixed abnormal	NS	*hypotrichotic* mixed abnormal	*hypotrichotic* active suspect
4	*normal* mixed abnormal	*normal* mixed abnormal	NS	*hypotrichotic* active n.a.d.	*hypotrichotic* mixed dysplastic
6	*normal.* mixed n.a.d	*normal* mixed n.a.d.	NS	*hypotrichotic* mixed n.a.d.	*normal* mixed suspect
7	*alopecic* mixed abnormal	*hypotrichotic* mixed abnormal	NS	*alopecic* mixed dysplastic	*alopecic* mixed abnormal
8	*alopecic* inactive dysplastic	*normal* inactive dysplastic	NS	a) *hypotrichotic* inactive dysplastic (b) *alopecic* inactive dysplastic	*hypotrichotic* mixed dysplastic
9	a) *hypotrichotic* mixed dysplastic (b) *hypotrichotic* mixed dysplastic	NS	NS	*hypotrichotic* mixed dysplastic	*hypotrichotic* mixed dysplastic
10	*hypotrichotic* inactive abnormal	*alopecic* inactive abnormal	NS	*alopecic* inactive dysplastic	*hypotrichotic* mixed suspect
11	a) *alopecic* mixed suspect (b) *alopecic* suspect	*normal* active suspect	NS	*alopecic* mixed suspect	*hypotrichotic* mixed suspect
12	*hypotrichotic* mixed suspect	NS	NS	*normal* mixed suspect	*hypotrichotic* mixed suspect
13	*hypotrichotic* mixed abnormal	*normal* mixed abnormal	NS	NS	*hypotrichotic* mixed abnormal
14	NS	a) *hypotrichotic* inactive dysplastic (b) *alopecic* inactive abnormal	NS	a) *hypotrichotic* inactive dysplastic (b) *alopecic* inactive dysplastic	NS
15	NS	*normal* active abnormal	a) *alopecic* inactive dysplastic (b) *hypotrichotic* inactive dysplastic	*alopecic* inactive dysplastic	NS
16	*hypotrichotic* mixed n.a.d.	*hypotrichotic* active n.a.d.	*normal* mixed n.a.d.	NS	*hypotrichotic* mixed suspect
17	NS	*normal* mixed abnormal	NS	a) *hypotrichotic* inactive dysplastic (b) *alopecic* mixed dysplastic	NS
18	*alopecic* mixed abnormal	a) *normal* mixed abnormal (b) *alopecic* mixed dysplastic	NS	*alopecic* mixed dysplastic	*alopecic* mixed abnormal
19	*hypotrichotic* inactive n.a.d.	*normal* active suspect	NS	*hypotrichotic* inactive dysplastic	NS
20	*normal* active suspect	NS	NS	*a) alopecic* inactive dysplastic (b) *hypotrichotic* mixed dysplastic	NS

NS: not sampled; **n.a.d**. no abnormality in morphology detected; **inactive**: no inferior follicles or active hair bulbs present; **active**: all follicle groups contain at least one growing hair; **mixed**: at least one follicle group contains at least one growing hair; **dysplastic**: dilated/keratotic infundibulum and increased small sebaceous lobules; **abnormal**: altered morphology consistent with mild pilosebaceous dysplasia; **suspect**: minimal alteration suggestive of very mild pilosebaceous dysplasia.

using standard histological methods. All sections were initially examined 'blind', i.e. without prior knowledge of the condition or site of the skin sampled. Results were subsequently reviewed in relation to the clinical findings.

Management. To reduce variability in management during these studies owners were asked to feed their dogs at the first examination, a balanced commercial dry diet containing fish and corn (Wafcol; Fish & Corn) (Table 2) with just water to drink. No dietary supplements were permitted. Routine flea control on dogs (Frontline spray, Merial) and in the environment (Acclaim 2000, Sanofi) was instituted in those cases in which it was not already in progress.

RESULTS

Responses to the questionnaire were obtained from 132 of the 216 IWSA members and covered a total of 146 dogs. Sixty-four owners had dogs without any cutaneous disorder, 41 reported having dogs with coat abnormalities and 27 had dogs with various types of skin lesions (Fig. 3). Twenty-four dogs were recruited and examined. There were 16 females (nine of which had been spayed) and eight males (two of which had been castrated). Their ages were between 1 and 11 years (mean 6.50 $\pm$ 2.99 standard deviation).

Clinical findings in dogs with coat disorders

General physical examination revealed no abnormalities. Dermatological examination revealed various patterns of alopecia or hypotrichosis of differing severity.

In affected animals more or less extensive focal areas of hair loss affected the ventral and lateral neck (12 cases), rump (17 cases), trunk (11 cases), or the thighs (14 cases) (Fig. 4a,b,c,d,e,f).

Pyoderma was a problem in 10 cases with the presence of numerous papules, pustules and epidermal collarettes, as well as comedones and varying degrees of scaling in the areas of hair loss, mainly on the dorsum or on the rump. In the hypotrichotic areas, the coat colour was lighter than in the other areas of the body and a high proportion of secondary hairs could easily be removed by stroking the coat.

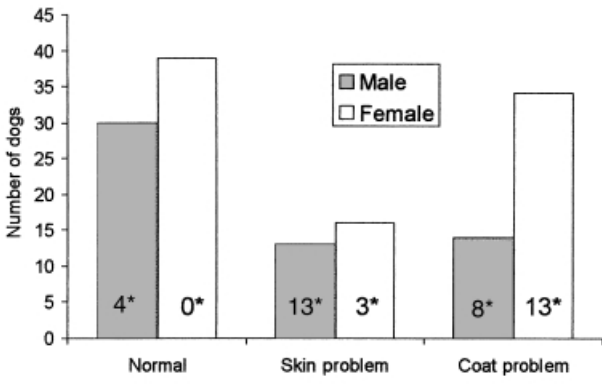

Figure 3. Numbers of dogs in each of the three categories derived from the epidemiological survey. (*number of neutered dogs).

All owners and/or breeders reported that their bitches developed hair loss in relation to the oestrus cycle. The initial focal hair loss had usually occurred on the rump and the thighs 6–8 weeks following the first or second oestrus cycle with subsequent hair regrowth 3–4 weeks later. A similar presentation occurred during subsequent oestrus cycles when the hair loss became more and more extensive, usually bilateral and well demarcated, and involving the lateral neck, rump, trunk or the thighs. After a few oestrus cycles the hair loss became permanent. The hair loss did not occur in two bitches which had been injected with progestagens but did occur again when their oestrus cycles were restored.

All males presented similar pattern of hair loss sometimes starting early in life (1–2 years of age) but more commonly later (5–6 years of age). In all cases there was no variation of the hair loss with season of the year and hair loss progressed with age.

Laboratory investigations

Four dogs were not included because pruritus was the predominant feature. Diagnoses in these animals were scabies (1), food (1) and flea bite hypersensitivity (2). In the other 20 cases, dermatological investigations failed to reveal any ectoparasites. Trichograms of the hairs, which were easily removed, showed that they were in the telogen phase. In two cases, numerous hairs had shaft abnormalities consistent with trichorrhexis nodosa (Fig. 5a,b); scanning electron microscopy of the abnormal hairs in a single dog confirmed the structural defect (Fig. 6). Fungal culture gave growth of *Microsporum canis* in cases 8 and 10, which were littermates and living in the same house.

Routine haematological and biochemical investigations gave values which were within normal limits apart from high cholesterol values in 5 cases (Table 1). Hormonal assays showed a low level of total T4 in 3 cases with a normal response after a TSH stimulation test in case 16 and below normal levels in cases 8 and 10, which were also affected by dermatophytosis. Slightly raised values for oestradiol in an entire male (case 3) and in a neutered male (case 16), and for progesterone in case 16, were present but increased concentrations of 17-OHP pre- (in four cases) and post-ACTH stimulation (in all cases except case 16) were also present (Table 1).

Histopathological findings

Twenty-two samples were from alopecic areas of skin, 33 from hypotrichotic areas and 19 from normal sites. Most sections gave reasonable vertical sections of the hair follicles with 2–7 follicle groups included per section. Each section was graded as active, mixed or inactive depending on the presence or absence of anagen follicles (Table 3). Occasional sections were oblique or almost transverse so that it was impossible to assess the inferior portion of the follicles and classifica-

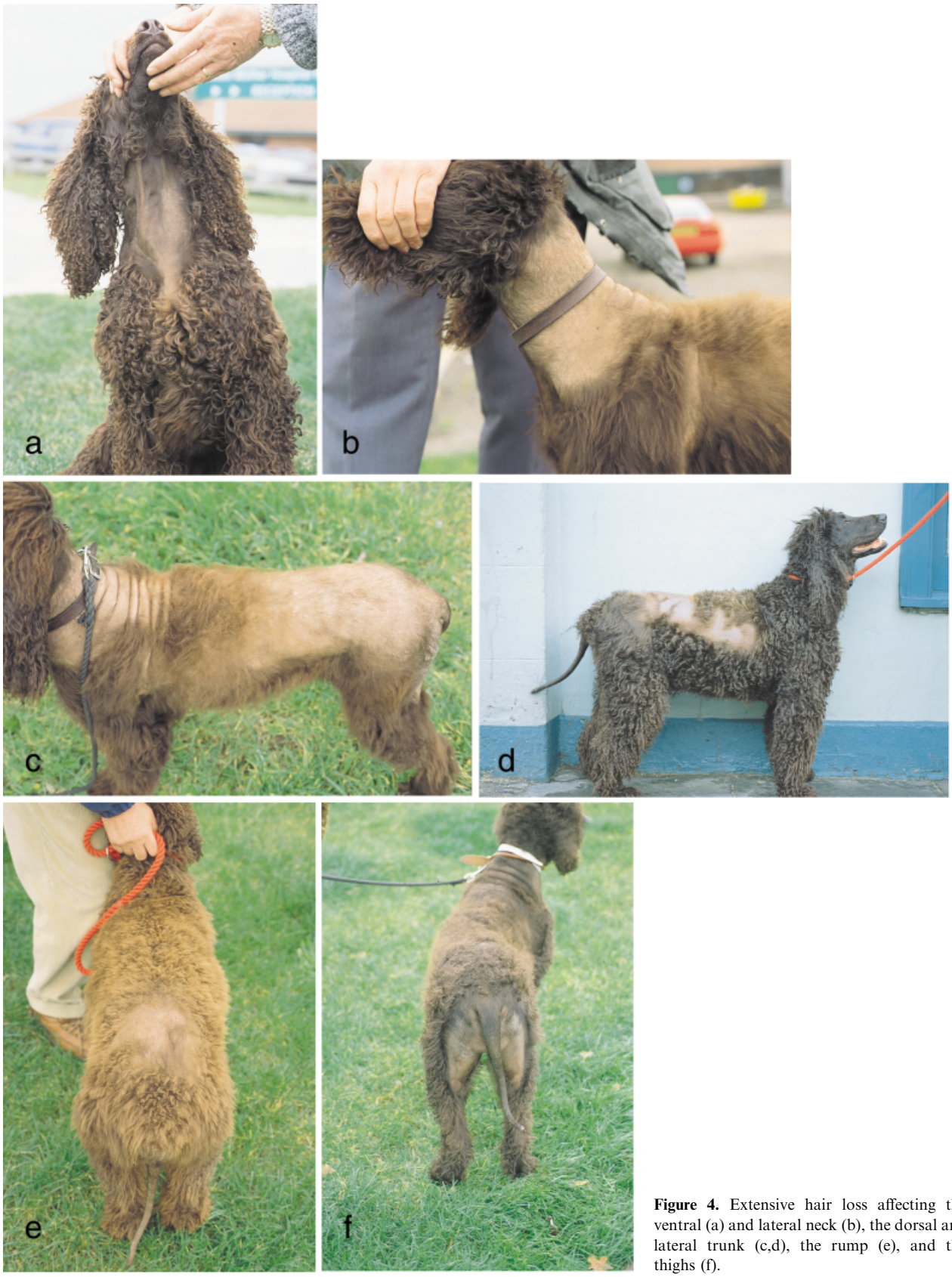

Figure 4. Extensive hair loss affecting the ventral (a) and lateral neck (b), the dorsal and lateral trunk (c,d), the rump (e), and the thighs (f).

tion depended on the presence of clearly identifiable inner root sheath in mid-dermal follicle segments.

Alopecic samples were characterized by diminished or absent follicular activity in most follicle groups and by the presence of aberrant pilosebaceous units in 55% of samples. In the most severely affected specimens (Fig. 7) pilosebaceous units were represented by dilated and keratotic follicle infundibula, often associated with increased numbers of small sebaceous lobules and below the atrophic remnants

of the lower follicle were present. These abnormal pilosebaceous units were recorded as dysplastic (Table 3). Similar but milder changes of infundibular dilatation and keratosis and increased sebaceous lobulation (recorded as abnormal) were seen in hypotrichotic samples and also as a more subtle alteration (recorded as suspect) in a few normal samples (Table 3). In 15 dogs (79%) there was at least one area of skin with inactive follicles and/or mild to marked alteration in pilosebaceous morphology.

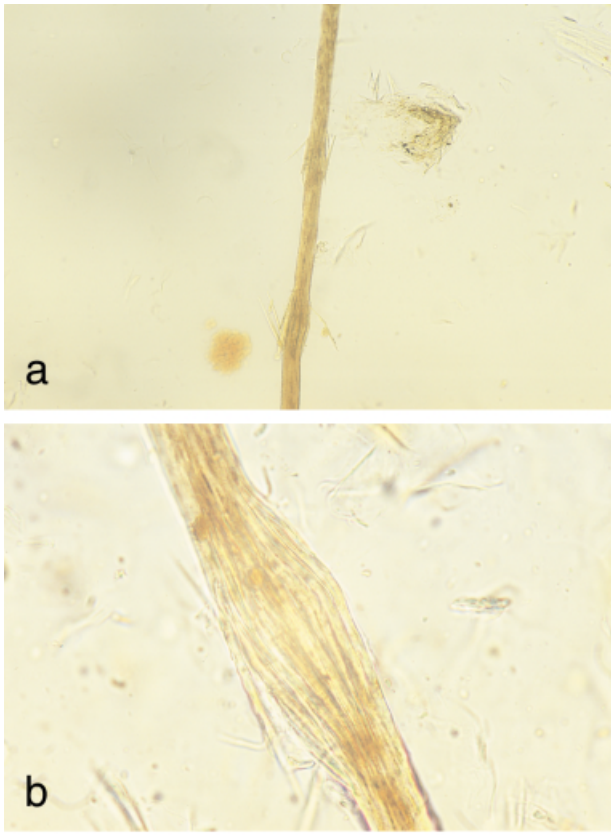

Figure 5. Direct microscopical examination of hairs (a, b) from case 5 showing foci of trichorrhexis nodosa (a: × 50; b: × 125).

An additional feature recorded in all dogs (85% of specimens) was clumped pigment in the hair follicle and/or the hair shaft (Figs 8 and 9). There was also some degree of pigmentary incontinence (Fig. 9) in the deep dermis and subcutaneous tissue in 88% of specimens, including all the normal samples. Pigmentation associated with the hair follicle was considered to be heavy in 14 samples (including 1 alopecic and 5 normal) but in no sample was pigment clumping observed in the epidermis. Sparse pigmentation of the sebaceous glands was present in 9 sections (5 alopecic, 4 hypotrichotic).

One or more flame follicles (Figs 9 and 10) representing catagen or early telogen follicles with excess trichilemmal keratin were seen in 22 sections including 5 from clinically normal skin.

Small fine hairs or small hair bulbs and tiny hair germs (Fig. 11) were noted in 38 samples from 17 dogs. These appearances were recorded in 9 (47%) of the normal skin samples as well as the 29 (53%) from affected skin areas.

Superimposed inflammatory lesions and/or epidermal thickening were noted in 6 of the 9 cases sampled which had a history of pyoderma and also in a further 5 dogs. These lesions were mainly focal and ranged from moderate to mild or, in some cases, minimal.

Management

In nine dogs with concurrent superficial pyoderma, *Staphylococcus intermedius* was isolated from the swabs submitted for bacteriological culture and was found to be sensitive to various antibiotics, including co-amoxiclav, cephalexin and enrofloxacin. One of these antibiotics was chosen and the therapy was carried out for at least four weeks.

Sixteen dogs were maintained on the fish and corn diet (Wafcol). Biochemical analysis of the essential fatty acids (EFAs) of the diet (fish and corn) used in this study shows that the ratio of omega 6 to omega 3 is 2.7: 1.

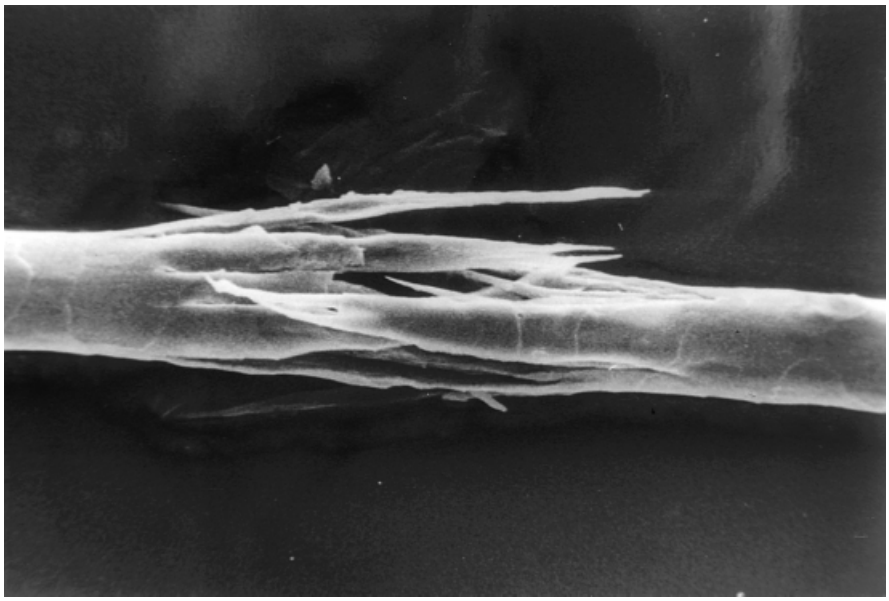

Figure 6. Scanning electron micrograph of an affected hair with damage to the hair shaft in trichorrhexis nodosa.

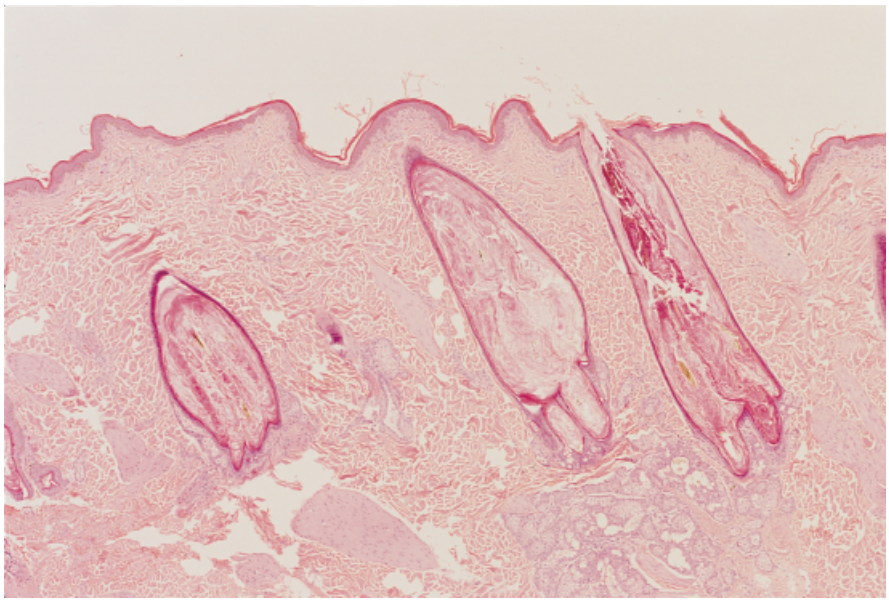

Figure 7. Sample of alopecic skin. Note small sebaceous lobules associated with dilated and keratotic follicle infundibula (H & E × 10).

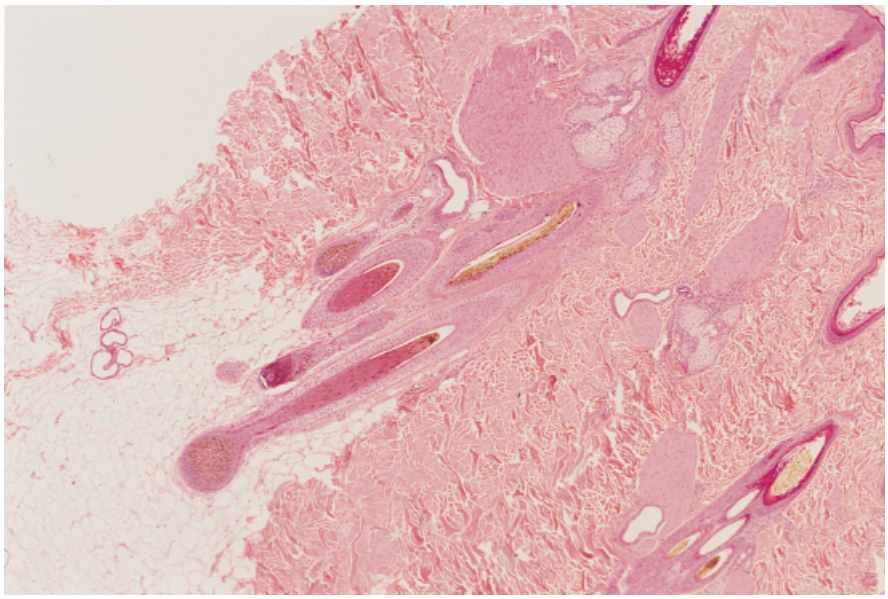

Figure 8. Anagen hair follicles in a sample of normal skin. Note pigment clumps in hair shaft (centre) and outer root sheath (top right) (H & E × 16).

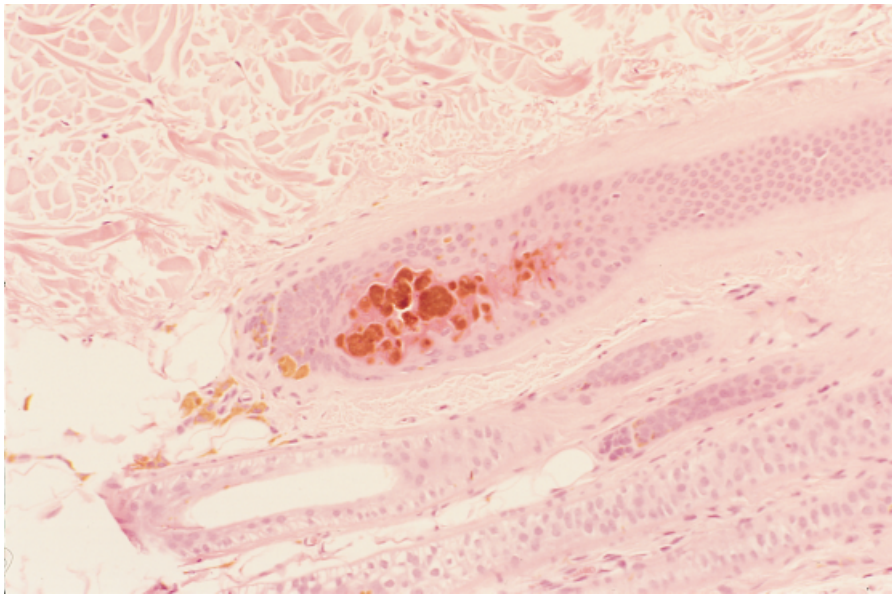

Figure 9. Hypotrichotic skin. Large clumps of pigment are present in a flame follicle. Note the pigmentary incontinence into the subjacent connective tissue (H & E × 50).

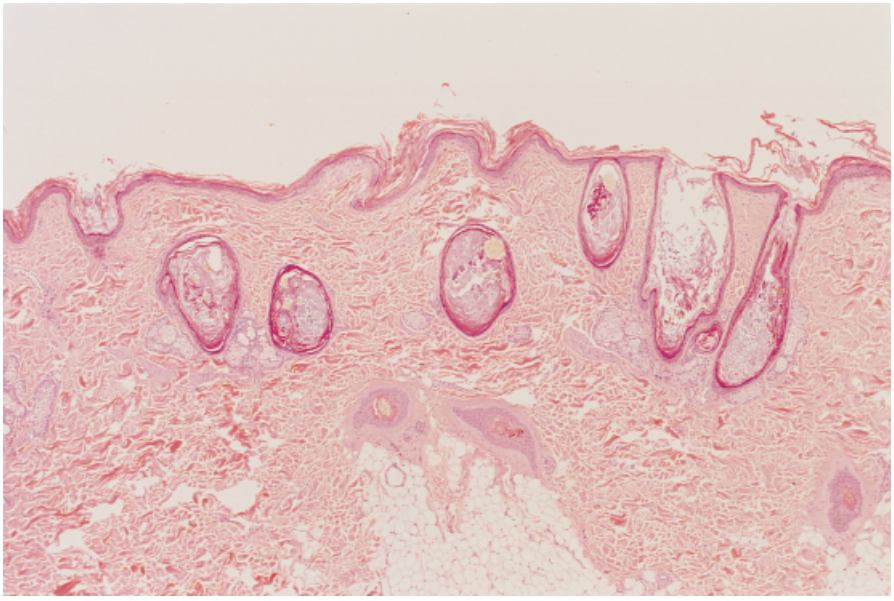

Figure 10. Hypotrichotic skin. Oblique profiles of two flame follicles are present (H & E × 10).

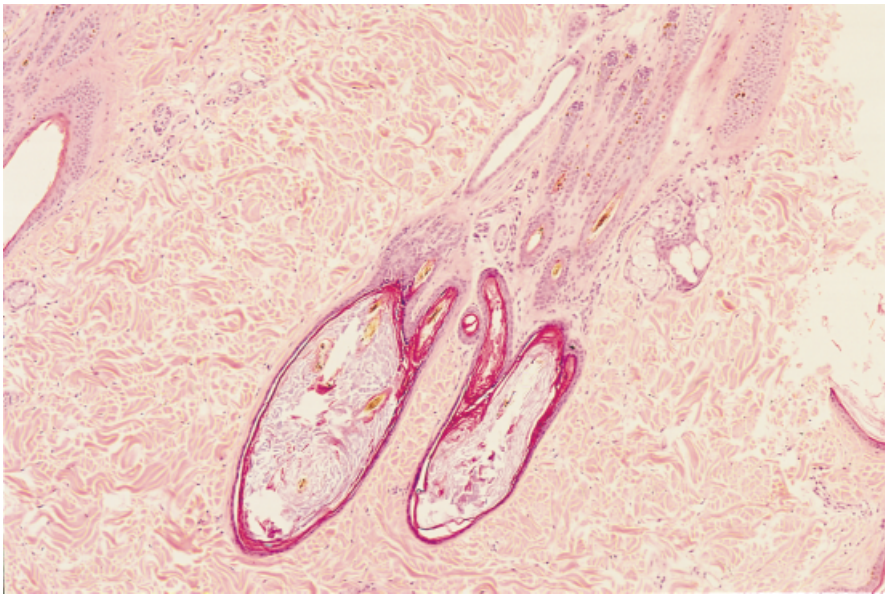

Figure 11. Hypotrichotic skin. Minute hair bulbs and developing follicles are present below dilated and keratotic infundibula (H & E × 20).

Thyroxin supplementation (20μg kg^{-1} twice daily) and griseofulvin (40 mg kg^{-1} daily for six weeks) therapy was started in the two hypothyroid dogs.

Follow-up

Routine re-examinations were carried out every 4–5 months over a period of one and half years. No relapse of infection was noted following the initial treatment, with antibiotic, of the dogs with pyoderma. No hair regrowth was observed at the site of the skin biopsy samples collected from the alopecic areas. Following the introduction of the fish and corn diet, a dramatic improvement of the skin and hair quality was observed in seven of 16 dogs. Three others showed localized and partial improvement whilst on the diet. Hair regrowth was more pronounced in some areas of the body than in others (Fig. 12a,b,c,d). Trichorrhexis nodosa was no longer present in the previously affected cases (3 and 5). In case 13 the coat had improved but changing the diet away from the fish and corn, following struvite crystals formation in the bladder, had resulted in coat deterioration. Restoring the diet resulted in an improvement of the coat condition again. Anecdotal reports in two cases in which the diet was supplemented with a combination of evening primrose oil and fish oil also confirmed improvement in the coat quality. The two dogs with hypothyroidism and dermatophytosis did not show any relapse of infection following griseofulvin therapy suggesting that both the antifungal therapy and the thyroxine supplementation had helped. Their post-pill concentration of total T4 was always within the normal range when monitored at six-month intervals. However these dogs have shown no hair regrowth after one year. Anecdotal reports by four owners who had previously treated their dogs with thyroxine supplementation, on the assumption of a suspected hypothyroidism, had not resulted in any hair regrowth.

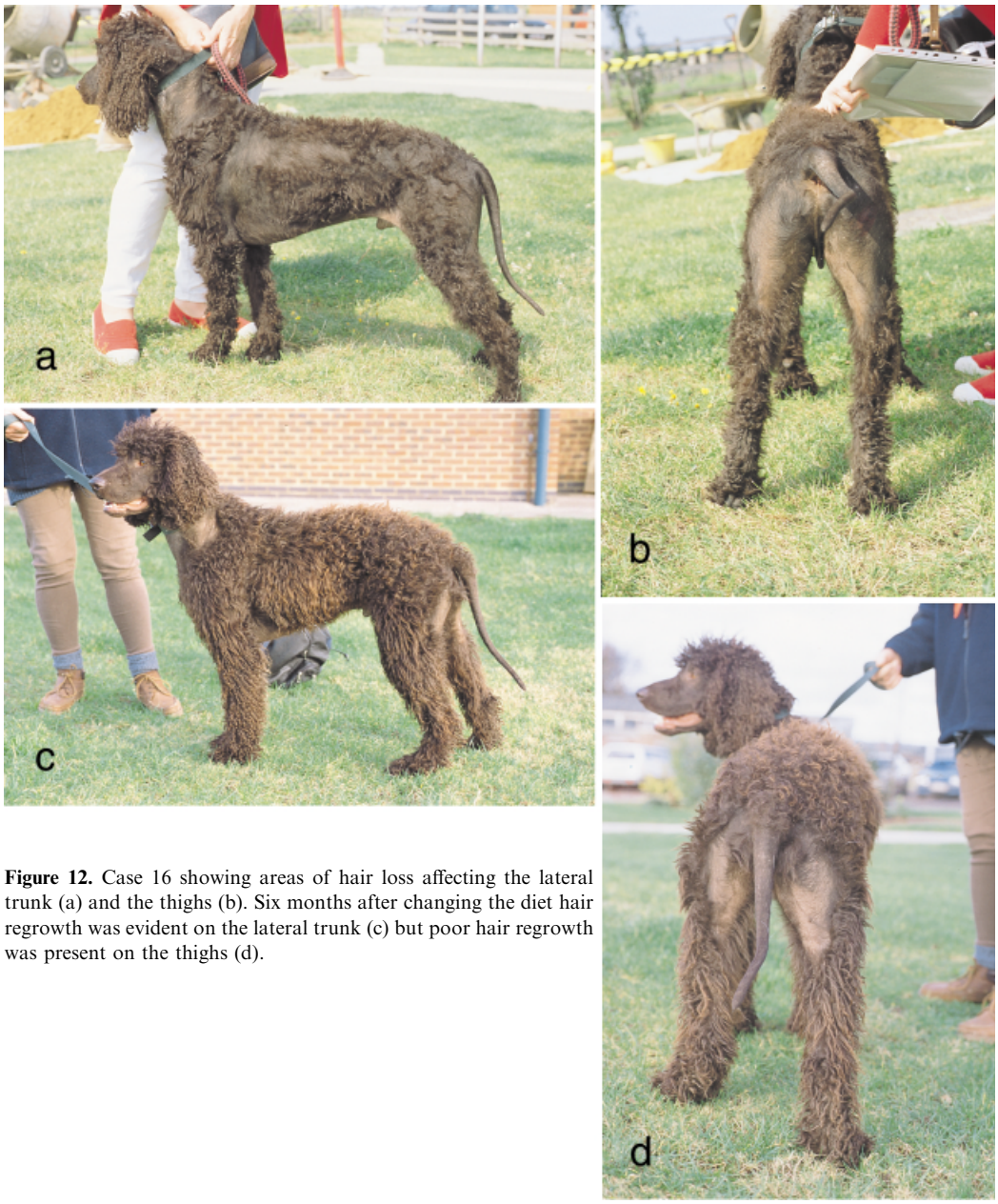

Figure 12. Case 16 showing areas of hair loss affecting the lateral trunk (a) and the thighs (b). Six months after changing the diet hair regrowth was evident on the lateral trunk (c) but poor hair regrowth was present on the thighs (d).

No improvement was seen in two of the dogs which only consumed the fish and corn diet for two months and then returned to their previous foods. One other dog has continued on the diet for over one year with no improvement. Two mildly affected dogs were reported to find the diet unpalatable. One returned to the previous diet with additional supplementation of essential fatty acids. The other had a succession of different diets. Both have shown coat regrowth. In two other cases seen within 4 months of starting the diet insufficient time has elapsed to determine whether improvement may occur.

Two dogs died of problems unrelated to the skin within one month of the first visit.

DISCUSSION

The coat problems affecting IWSs have caused a great deal of confusion among breeders and veterinary surgeons for many years. The occurrence of focal or diffuse areas of hair loss has been often misdiagnosed as an endocrine disorder or as dietary insufficiency with subsequent hormonal and/or vitamin and mineral supplementation.

The current study has enabled the clinical patterns, hormonal status and histopathology of this condition to be described for the first time. Previous reports have classified the hair loss affecting the caudomedial thighs as 'pattern baldness'[5,6] or have referred to the hair loss affecting the caudal dorsum and the lateral sides of the trunk as a follicular dysplasia.[5,7]

Pattern baldness is a term used in human dermatology, to illustrate the hereditary male or female type of androgenetic alopecia. It is characterized clinically by a progressive frontal and occipital hair loss. Histologically there is moderate to severe decrease in size (miniaturization) of the hair follicles, which are both shorter and thinner than normal, and

the residual hair shafts are thin. The adnexal structures are normal. A similar histological abnormality has been reported to occur in some canine breeds, although a different clinical presentation occurs. A progressive, usually bilateral, alopecia affects the ear pinnae or caudomedial thighs but it may also be noted on the chest and abdomen. This syndrome has been called canine pattern baldness and three different forms have been recognized and classified by Scott *et al.*[5] according to the affected breed and the clinical presentation. In some cases a genetic predisposition has been suspected.

Follicular dysplasia has been reported in many canine breeds and has been divided histologically into categories in which the hair cycle is abnormal (follicular dysplasia in the Siberian Husky, CRFA)[8,9] and those in which there are abnormalities in the process of melanization of the pilosebaceous units (colour dilution alopecia, follicular dysplasia of the Portuguese water dog).[10,11] Clinical signs and the localization of hair loss follow different patterns according to the aetiology.

In our study, affected dogs seem to show clinically different patterns of hair loss that are similar to both canine pattern baldness and follicular dysplasia of the Portuguese water dog.

Alopecic and hypotrichotic skin of the IWS shows follicular keratosis and atrophic follicles as seen in CRFA,[9,12] and also shows abnormal clumping of melanin and dystrophic hair follicles as seen in colour dilution alopecia.[12,13] However in IWS the condition is not seasonal and the dogs do not have the epidermal pigmentation pattern of the dilute coat colours. The presence of follicles showing excessive trichilemmal keratinization in skin from both normal and affected sites probably reflects either a form of hair cycle arrest or a breed tendency to form these flame follicles rather than any specific endocrine abnormality.[7] The presence of small fine hairs in up to half of the sites sampled is likely to be the result of loss of primary or guard hairs in a breed with numerous small secondary hairs, although no samples from clinically normal dogs were available for comparison. This loss of primary hairs has been reported previously in the IWS and also in the Portuguese water dog and curly coated Retriever which are believed to be afflicted by a similar form of canine follicular dysplasia.[7] It is interesting to note that Gross *et al.*[7] and Miller[9] have observed features of colour dilution alopecia in the Portuguese water dog while lesions in the IWS are reported by Gross *et al.*[7] as subtle alterations including lack of normal cycling and mild distortion of follicles. Further categorization of this disorder in the IWS requires histological evaluation of normal skin from the same sites in age and sex matched controls from the same breed.

Superficial pyoderma is a common finding in dogs with follicular dysplasia possibly caused by the abnormal hair follicles which predispose them to bacterial colonization. The presence of pyoderma in 10 dogs in this study may have contributed to the hair loss but when these concurrent conditions had previously been treated normal coat condition was never achieved.

Trichorrhexis nodosa (TN) is a hair-shaft abnormality in which the individual cortical cells and their fragments splay out and fray, resembling the ends of two paint-brushes pushed into one another. It may be evident with the naked eye but light and scanning electron microscopy will confirm the lesion.

In man this condition may be congenital or acquired. The acquired form is mainly caused by trauma but a contributory factor is inherent weakness of the hair shaft. Trauma such as excessive brushing, stressed hair styles, heat, ultraviolet exposure, trichotillomania, pruritus or chemical trauma such as salt water bathing, shampooing, perming and dyeing of the hair are the commonest causes. Underlying hair structural abnormalities such as a cuticular weakness and/or a cortical deficiency cause brittle hairs, which may predispose to TN. If the cuticle is damaged or destroyed, the cortex is exposed to more physical trauma and to detergents that have a solubilizing effect on the intercellular cement substance. Cortical deficiency includes a breakdown of the intercellular cement substance that holds the hair cortex cells together.[14]

In animals, the congenital form of TN has not been reported and the acquired form is uncommon.[15]

In this study TN was found in only two cases in which there was no history of chemical or irritant shampoo, of improper grooming or of ectoparasite infestation. The concurrent presence of superficial pyoderma and pruritus in one case with associated abnormal fragile hair due to follicular dystrophy may explain this finding. Both dogs showed resolution of the TN and hair regrowth following the dietary change and antibiotic therapy in one case. This suggests that amelioration of the follicular function may have reduced the fragility of the hairs.

The fact that pyoderma, which had previously been a recurrent problem, no longer occurred and coat normalization was observed in 7 of 16 dogs after institution of the standard diet implies that diet is a significant factor. The reason for this improvement is not clear. EFA content or other factors in the diet may have contributed to improved hair follicular function but further studies are necessary to elucidate the effect of diet in this condition. Although there was a marked improvement in most of the dogs, the hair regrowth did not occur in some areas, such as the lateral neck and the thighs, possibly because these areas are genetically predisposed to alopecia and other factors are involved in the hair loss.

In this preliminary study we evaluated the function of the thyroid and adrenal glands, and sexual hormonal status. Growth hormone (GH) production was not evaluated. Although GH responsive alopecia has been reported in a few cases,[16] it is likely that the hair regrowth, following GH supplementation, is not

caused by a primary GH deficiency but the GH is merely an additional stimulus to the function of the hair follicle. The circadian rhythm of this hormone and the variability of the results which may occur, as reported in the Pomeranian, and the difficulty in finding a laboratory for its assay makes its measurement difficult to carry out and its values difficult to evaluate, not justifying this investigation in our preliminary study.[17,18] GH therapy was not tried in the current study as the histopathology findings were not suggestive of such an endocrinopathy and the risk of side-effects using the above drug is high and the response to the therapy uncertain.

The thyroid function was checked by both endogenous TSH and total T4 assay and this combined determination has also been reported to be a useful indicator by Peterson.[19] A TSH stimulation test was carried out only in the few cases where a low basal total T4 and/or a high cholesterol level were found; hypothyroidism was confirmed in two cases, both of which also had dermatophytosis. It is unlikely that these conditions contributed to the observed pattern of hair loss, as there has been no hair regrowth since the T4 supplementation and the antifungal therapy, over one-year follow up, were commenced.

Values of cortisol pre- and post-ACTH stimulation test were shown to be in the normal range and furthermore no haematological and biochemical abnormalities, or clinical signs were present at the time of the first consultation or during one and half year follow up in affected dogs suggestive of hyperadrenocorticism. The increased levels of endogenous ACTH in two of the seven cases in which it was measured was not associated with other clinical signs or laboratory findings suggestive of hyperadrenocorticism.

The cyclic episodes of hair loss in the bitches support a cause and effect relationship between oestrus and alopecia. In bitches at the end of the pro-oestrus and the beginning of the oestrus there is a secretion of LH with subsequent increase of oestrogens, androstenedione and testosterone.[20] It is likely that the increase of such hormones or the presence of a high number of hormonal receptors at the level of the skin may be a stimulus for the hair follicle to pass from the anagen to the telogen phase with clinically evident hair loss 6–8 weeks later. The subsequent hair regrowth, clinically evident after a further 6–8-week period, may be the consequence of the reduction of these hormones and the rise of the progesterone levels at the end of oestrus. Another possible explanation of the alopecia is that oestrogen secretion occurring during the oestrus cycle is responsible for increased hormonal sensitivity at the level of the oestrogen receptors in the skin causing localized hair loss.[21] The hair loss and subsequent hair regrowth are not related to season as they can occur any time during the year, thus excluding conditions such as CRFA.

The number of affected males (entire and neutered) was insufficient to demonstrate a correlation between hair loss and sexual hormones. Castration in affected IWS does not seem to arrest the progression of the hair loss as occurs in canine castration responsive dermatosis and in humans with androgenetic alopecia.[22,23] Further studies are necessary to evaluate this.

The occurrence of alopecia which is more marked in females each time they have an oestrus cycle or in males after few years may suggest that there is an increased sensitivity of the hair follicle to the sexual hormones with advancing age as occurs in human beings with androgenetic alopecia.[24] The presence of hormonal receptors in histopathological sections of the skin in these dogs is currently under evaluation and the results will be published elsewhere. The time of onset of alopecia supports that there is a chronobiological clock regulating the process of alopecia in IWSs as reported in man[25] but this requires further investigation.

In human beings, the male and female types of pattern baldness, which start at puberty in the male or following hormonal changes in women are associated with increased formation of dihydrotestosterone and the presence of high levels of hormonal receptors at the level of the hair follicle in affected areas.[26] Castration in men, and use of anti-androgens or 5 α-reductase inhibitors in both sexes have confirmed that individuals, although genetically predisposed, show arrest of the progression of the baldness with subsequent hair regrowth.[23,27] Whether the same pathogenetic mechanism occurs in the IWS needs further investigation but it is likely that the high level of progesterone present after the oestrus cycle or the effect of the progestagen injections in bitches may contribute to a reduction in the transformation of testosterone to dihydrotestosterone, as pregnenolone acts as a competitive substrate with the testosterone, utilizing the same enzyme (5 α-reductase) with formation of 5 α-pregnanedione instead of dihydrotestosterone (Fig. 13).[28] Whether a high level of dihydrotestosterone and the presence of 5 α-reductase isoenzymes at the level of the hair follicle and adnexal structures are present in this canine condition needs to be further evaluated before this pattern of alopecia can be compared with human androgenetic alopecia.

The pre-and, in particular, post-ACTH stimulation concentrations of 17-OHP were increased in most of the cases in the current study. This hormone was also measured in three other IWSs affected by other cutaneous conditions and found to be within the normal range, which was established in a group of dogs of various breeds and measured by the same laboratory.[29] The determination of this hormone has been claimed as an indicator to assess adrenal and gonadal steroidogenesis. A high concentration of 17-OHP which may be associated with high concentrations of progesterone, androstenedione, testosterone and endogenous ACTH are indicative, in man, of congenital adrenal hyperplasia (CAH). CAH is caused by the lack, or partial deficiency, of either 3 β-hydroxysteroid dehydrogenase, 21-hydroxylase or

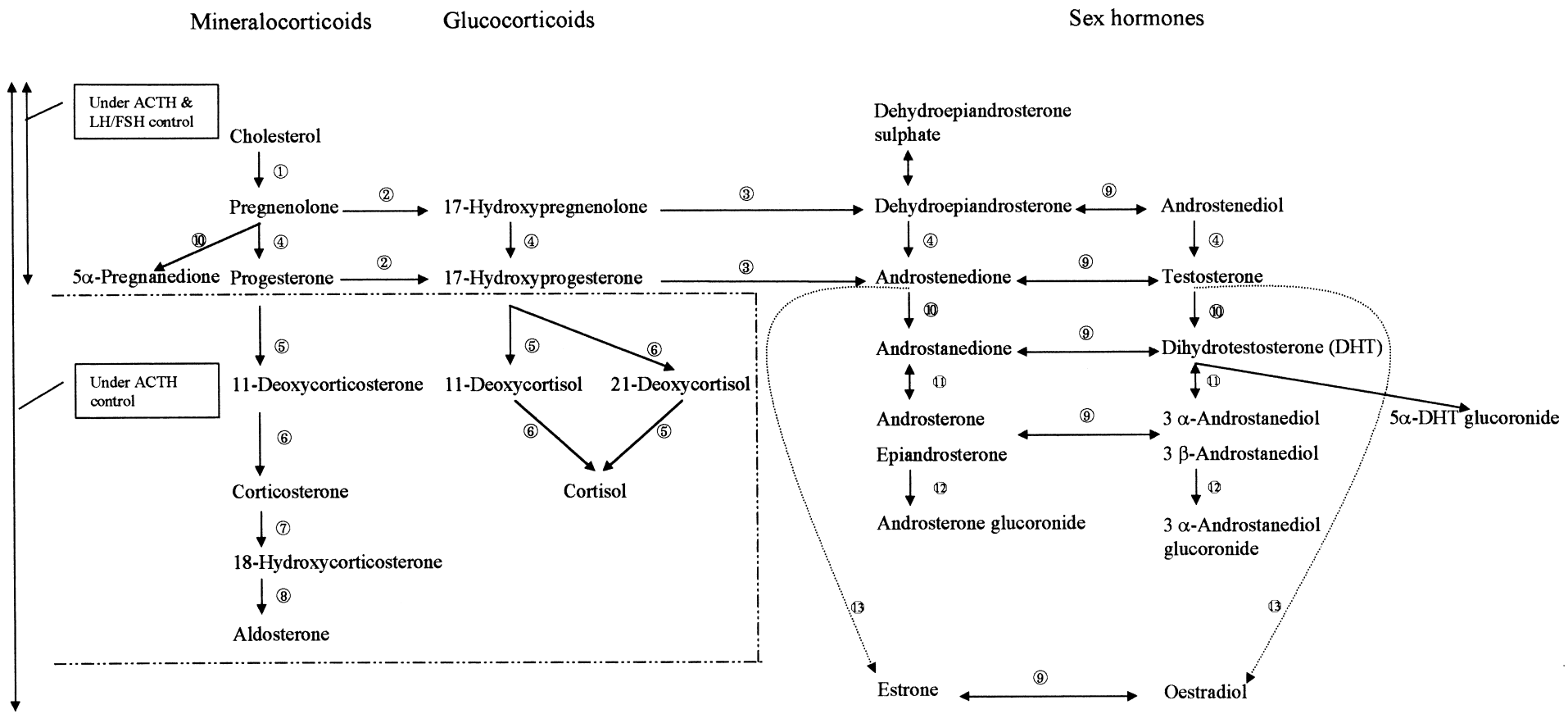

Figure 13. Adrenal and gonadal steroidogenic pathways. The lack of either 21-hydroxylase or 11 β-hydroxylase is a common cause of congenital adrenal hyperplasia in man. (1) Side-chain-cleavage cytocrome P450; (2) 17 α-hydroxylase P450; (3) 17–20-Lyase P450; (4) 3 β-Hydroxysteroid dehydrogenase $\triangle^5$– $\triangle^4$-isomerase; (5) 21-Hydroxylase P450; (6) 11 β-Hydroxylase P450; (7) 18 β-Hydroxylase; (8) 18-OH dehydrogenase; (9) 17 β-Hydroxysteroid dehydrogenase; (10) 5 α-reductase (type I and II); (11) 3 α- or 3 β-Hydroxysteroid dehydrogenase; (12) β-glucoronidase; (13) Aromatase P450.

11 β-hydroxylase (Fig. 13).[30–32] This condition has been suspected in breeds such as the Pomeranian and American Eskimo dog.[33,34]

In our cases, the presence of bilaterally symmetrical alopecia with hair discoloration and variable degrees of seborrhoea, and the increased concentrations of 17-OHP, points to a condition similar to that reported in the Pomeranian. The hyperpigmentation and the hair regrowth at the site of the skin biopsy, common in Pomeranian, are not present in these cases. Further studies are necessary to evaluate the other hormones involved in the steroidogenic pathways either in affected and in normal IWSs so the defect can be identified and characterized.

The concentration of endogenous ACTH, which may be high in cases of CAH, has been measured only in seven cases and found to be high in two of them, suggesting possible CAH, although episodic fluctuations of ACTH levels have been reported in normal dogs.[35] Therefore multiple sampling may be necessary to evaluate the ACTH production and further determinations in the other affected dogs will be necessary. It is worth noting that in mild, nonclassical forms of CAH in man, the concentrations of endogenous ACTH are commonly reported to be within the normal range[36] and this may be the case in the dog.

In man, the therapeutic approach to CAH involves the use of corticosteroids, while in dogs, adrenolytic drugs such as mitotane have been suggested.[18,31,34] A trial therapy in our cases, to suppress the adrenal cortex production of sexual hormones with corticosteroids or adrenolytic drugs, has not been carried out as it is considered premature until the possibility of CAH is evaluated.

Further investigations are in progress to study other hormones and their urinary metabolites involved in the canine steroidogenic pathway. This will rule in or out the possibility of a form of CAH-like syndrome in this breed.

ACKNOWLEDGEMENTS

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Résumé Cette étude s'est intéressée à la fréquence des dermatoses chez les Irish Water Spaniel (IWS) au Royaume-Uni. Un groupe de 20 chiens a été sélectionné et a subi un examen clinique. Tous les chiens présentaient une chute de poils localisée, non prurigineuse, non-inflammatoire, affectant les mêmes zones chez les mâles et chez les femelles, bien qu'une distribution cyclique associée au cycle oestral ait été observée initialement. Des examens hormonaux ont montré des signes compatibles avec une anomalie de la stéroïdogénèse. L'examen histopathologique a montré des images semblables à celles observées dans l'alopécie cyclique des flancs (CRFA) et de dysplasie folliculaire associée à une mélanisation anormale, comme en cas d'alopécie des robes diluées, bien que les signes cliniques ne correspondent pas à ces maladies. Des modifications alimentaires ont permis une amélioration de la qualité de la peau et du pelage dans la plupart des cas de cette série, mais le rôle précis joué par l'alimentation n'a pas été exploré plus précisément. Cette étude suggère que la chute de poils chez les IWS est influencée par des facteurs alimentaires et les hormones sexuelles. Des anomalies dans la synthèse des stéroïdes pourraient contribuer à la gravité de la maladie. [Cerundolo, R., Lloyd, D. H., McNeil, P. E. et Evans, H. An analysis of factors underlying hypotrichosis and alopecia in Irish Water Spaniels in the United Kingdom. (Analyse des facteurs responsables de l'hypotrichose et de l'alopécie chez les Irish Water Spaniels au Royaume-Uni.) *Veterinary Dermatology* 2000; **11**: 107–122.]

Resumen Se llevó a cabo en el Reino Unido un estudio de la presencia de dermatosis en el Irish Water Spaniel (IWS). Se seleccionó y examinó clínicamente un grupo de 20 perros. Todos los perros mostraban una pérdida de pelo localizada, no-prurítica y no-inflamatoria, afectando las mismas áreas corporales en machos y hembras, aunque se identificó un patrón cíclico inicial asociado al ciclo estral. Las investigaciones hormonales mostraron características sugestivas de una anormalidad en la esteroidogénesis. La histopatología reveló características similares a la alopecia recidivante de los flancos (CRFA) y una displasia folicular asociada con melanización abnormal, como en la alopecia de color diluido, aunque las características clínicas no se correlacionaban con estas dermatosis. Los cambios en la dieta mejoraron la calidad del pelaje y de la piel en la mayoría de los casos estudiados, pero no se investigó en profundidad el papel de la dieta. Este estudio sugiere que la pérdida de pelo en el IWS está influida por factores de la dieta y por las hormonas sexuales. Las anormalidades en las vías de esteroidogénesis pueden contribuir a la severidad de esta dermatosis. [Cerundolo, R., Lloyd, D. H., McNeil, P. E. y Evans, H. An analysis of factors underlying hypotrichosis and alopecia in Irish Water Spaniels in the United Kingdom. (Análisis de factores subyacentees a la hipotricosis y alopecia de los Irish Water Spaniels en el Reino Unido.) *Veterinary Dermatology* 2000; **11**: 107–122.]

Zusammenfassung Im Vereinigten Königreich wurde eine Umfrage über das Vorkommen von Dermatosen bei Irischen Wasserspaniels (IWS) durchgeführt. Eine Gruppe von 20 Hunden wurde ausgewählt und klinisch untersucht. Alle Hunde zeigten nichtjuckenden, nichtentzündlichen, lokalen Haarausfall, der bei männlichen

und weiblichen Tieren dieselben Körperstellen betraf, obwohl anfänglich ein mit dem Östrus assoziierter zyklischer Verlauf festgestellt wurde. Hormonelle Untersuchungen zeigten auf abnormale Steroidsynthese hindeutende Charakteristika. Histopathologie ergab Befunde, die der kaninen rezidivierenden Flankenalopezie und follikulären Dysplasie glichen, verbunden mit abnormaler Melanisierung wie sie in 'Colour dilution alopecia' gesehen wird, obwohl die klinischen Befunde nicht mit diesen Erkrankungen korrelierten. Fütterungsänderungen verbesserten die Haut-und Haarqualität in den meisten dieser Fälle, aber die Rolle, die das Futter spielte, wurde nicht weiter untersucht. Diese Studie deutet darauf hin, dass Haarausfall bei IWS durch Fütterung und Sexualhormone beeinflusst wird. Abnormale Steroidsynthese mag zu der Schwere der Erkrankung beitragen. [Cerundolo, R., Lloyd, D. H., McNeil, P. E. und Evans, H. An analysis of factors underlying hypotrichosis and alopecia in Irish Water Spaniels in the United Kingdom. (Eine Analyse der dem Haarmangel und der Haarlosigkeit der Irischen Wasserspaniels zugrundeliegenden Faktoren im Vereinigten Königreich.) *Veterinary Dermatology* 2000; **11**: 107–122.]