placenta is associated with a lower bodyweight. No other significant associations were identified. It is also considered that the work involved in examining the liver of calves dying around the time of calving is not justified as a means of diagnosing leptospiral infection if the lung, adrenal gland and kidney are available; however, the submission of placenta from full-term calves should be encouraged if leptospirosis is being investigated.

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Studies on the inheritance of hair loss in the Irish water spaniel

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The inheritance of hair loss in Irish water spaniels has been studied by evaluating the inbreeding levels and genetic relationships in a group of affected and unaffected dogs. A detailed study of the pedigree tree of four families revealed a familial predisposition suggesting a dominant mode of inheritance.

THE Irish water spaniel (IWS) is a breed with peculiar coat characteristics. The breed standard states that an area of smooth hair forming a V-shaped patch should be present on the throat, and that the tail should be bare (Anon 1996). For many years the breed has been selected for this pattern which is clearly an abnormality for any breed of dog. In recent years differing patterns of alopecia and/or hypotrichosis affecting variously the laterodorsal aspect of the neck, flanks, dorsum, rump and the caudal part of the thighs have been reported in the breed (Kummel 1990, Lee Gross and others 1992, Griffin 1993, Scott and others 1995). Since 1995 the authors have collaborated with the British IWS Association to characterise and investigate the causes of these coat problems. A postal survey was carried out among the members of the association and alopecic dogs were examined. When they could not be examined directly, relevant information was provided by the attending veterinarian. Routine dermatological investigations with skin biopsy, blood investigations (including hormonal tests), and dietary studies were carried out in the affected dogs (Cerundolo and others 2000). This work indicated that histopathological abnormalities of the hair follicle are common in the breed and are influenced by other variables. Gender differences are apparent in that susceptible females invariably develop the alopecia by two years of age, in association with the oestrus cycle, whereas males, if susceptible, may not show any changes until they are five or six years old. In addition, dietary changes may accentuate or diminish the signs of alopecia. These factors and their variability are likely to make it difficult to interpret the results of any study of the aetiology of the condition. Nevertheless a preliminary genetic analysis, with the aim of characterising the mode of inheritance of alopecia in the breed, has been attempted on the basis of a breed survey and a more detailed examination of the ancestry of four families of alopecic dogs.

MATERIALS AND METHODS

Data collection

From the survey, information was particularly sought on family data. The dogs were divided into three groups: affected, not affected and of unknown disease status, a group in which, for example, animals had died prematurely, or could not be traced, or were too young for a definitive diagnosis. The affected dogs had histories, clinical signs and pathological features consistent with those observed by the authors previously. When the dogs could not be examined directly, a full veterinary history, including the results of dermatological examinations and diagnostic procedures, clinical photographs and pedigrees, was obtained. Five-generation pedigrees of 10 affected dogs and 10 unaffected control dogs were obtained. Because the affected dogs commonly had affected littermates, these pedigrees effectively represented 10 litters with and 10 litters without affected individuals. The control dogs were chosen to be age matched (within six months) and without regard to whether any ancestors had been affected; they were

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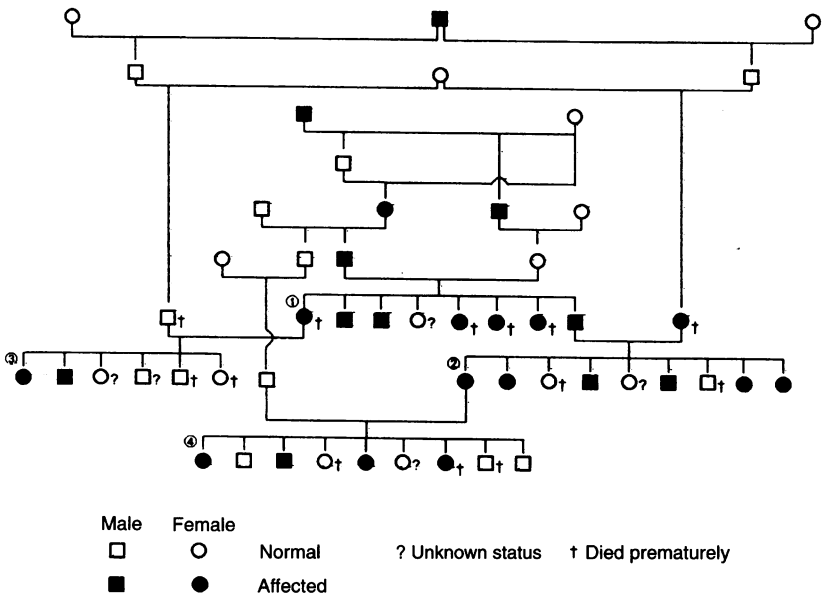

FIG 1: Pedigree tree of the four families studied in detail. Circled numbers indicate the four family groups

selected by one of the authors (R. C.) from among dogs attending two IWS shows, at Birmingham and Nottingham.

Inbreeding levels

The inbreeding coefficient (F) of each of the 10 affected and 10 control dogs was calculated from the formula of Wright given by Falconer (1989):

$$F_c = \Sigma(\tfrac{1}{2})^{n_1+n_2+1}.(1+F_a)$$

where F_c is the inbreeding coefficient of the individual dog, Σ indicates summation in case of multiple common ancestry, $^{n}_{1}$ is the number of generations between the sire and the common ancestor, $^{n}_{2}$ is the number of generations between the dam and the common ancestor, and F_a is the inbreeding coefficient of the common ancestor. The distributions of the inbreeding levels in the affected and the control groups were compared by using a Mann-Whitney non-parametric test appropriate for the comparison of inbreeding coefficients.

Genetic relationships

An estimate of the relative contribution of 10 commonly occurring ancestors (identified visually from the pedigrees) was made to see whether specific ancestors were implicated in a quantitatively different way in affected and unaffected dogs. Thus, for each ancestor, the number of times it occurred in the five-generation pedigrees of both the affected and unaffected dogs was counted. For the 10 ancestors relative risks were calculated as:

$$ad/bc$$

where a is the number of occurrences of a particular ancestor in the affected pedigrees, b is the number of occurrences in the control pedigrees, c is the number of cases without this ancestor, and d is the number of controls without this ancestor (Peeters and Ubbink 1994).

Family data

The ancestry of four litters containing affected dogs was also studied in detail in an attempt to identify any genetic determinants. A composite pedigree was constructed for this purpose.

RESULTS

Inbreeding level

The inbreeding coefficients of the 10 affected and 10 control dogs showed that there was no significant difference between the mean values of the two groups (Table 1).

Genetic relationships

Three ancestors were identified as having a high relative risk. All the other ancestors had a relative risk close to 1 (Table 2).

Family data

Fig 1 shows a composite pedigree of four families with affected members. It contains 37 dogs (16 males and 21 females) of which 21 were affected, five were not affected, five were of unknown status and another six had died prematurely. Of the affected dogs, 15 were examined directly. Eight males and 13 females were affected. It is apparent that consanguinity levels were relatively high. A strong familial pattern of transmission is evident, affected individuals, almost invariably, had at least one affected parent, which also had an affected dam, sire or both. Segregation analysis was not possible because too many of the dogs were of unknown status.

The pedigree data suggest that a single autosomal gene may play a major causative role in this abnormality, and the transmission pattern provides evidence for a dominant mode of inheritance.

DISCUSSION

Purebred dog populations represent closed gene pools and a relatively high level of consanguinity and as a result inbreeding is common (Patterson and others 1989, Willis 1989). The finding of a common ancestry among affected dogs does not necessarily indicate the genetic involvement of these common ancestors in the spreading of a disease through the population,

TABLE 1: Coefficients of inbreeding (F) of the 10 affected and 10 control dogs

	Affected	Normal
1	13·67	9·57
2	11·72	20·70
3	0·78	8·78
4	0	10·35
5	8·11	3·91
6	3·52	2·73
7	10·16	2·93
8	4·50	3·91
9	8·20	0
10	16·41	14·06
Mean	7·70	7·69

TABLE 2: Relative risks for 10 commonly occurring ancestors in affected and normal Irish water spaniels

Ancestors	Sex	Relative risk
A	M	0·87
B	F	1·71
C	F	1·25
D	M	0·61
E	M	11·25*
G	M	9·0*
H	M	23·4*
I	M	1·20
J	M	2·29

*High relative risk

because the common ancestors could be a characteristic of the population rather than a characteristic of the affected dogs.

Inbreeding is not responsible for genetic disease. Some degree of inbreeding is necessary to develop homozygosity for both desirable recessive and desirable dominant genes and the consequent phenotypic uniformity within a breed or line. In fact selection within a small population for a desired genetic trait usually requires a degree of inbreeding. The levels of inbreeding observed in this study of IWS were within expected limits, and there was no difference between the levels of inbreeding of the small groups of affected and control animals.

The genetic relationships investigated did show a high relative risk for three specific ancestors, which suggested that these ancestors transmitted a gene or genes which increased susceptibility to hair loss. The nature of this genetic determination is indicated to some extent by the pedigree information which shows strong familial transmission and a possibly dominant mode of inheritance. It is not possible to determine the precise mode of inheritance, but the gene(s) responsible must be relatively common in this part of the population. The fact that there was no difference between the mean inbreeding levels of the affected and unaffected dogs favours a dominant hypothesis. The data are not entirely compatible with a single gene model. It is possible that a single major gene is segregating and interacting with some minor susceptibility variants. It can be reasonably concluded that these minor and major genes have been accumulated through the selection for the peculiar and specific pattern of smooth throat and bare tail desired in the breed.

This genetic predisposition to alopecia seems to be linked to abnormalities of the steroidogenic pathway and, in particular, to increased levels of 17-hydroxyprogesterone before and/or after an adrenocorticotrophic hormone stimulation test (Cerundolo and others 2000). This pattern has some similarities with congenital adrenal hyperplasia in man (Pang 1997), which is also a genetically programmed disease in which measurements of urinary steroids are used as a diagnostic tool.

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Use of a balloon occlusion catheter to facilitate transarterial coil embolisation of a patent ductus arteriosus in two dogs

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Two dogs with a history of coughing and exercise intolerance were suspected to have a patent ductus arteriosus (PDA), and the presence of a type III PDA was confirmed by radiography, electrocardiography, ultrasonography and angiography. Transarterial coil embolisation was carried out by using a modified technique. An occlusion balloon catheter was inserted through a femoral vein and placed at the pulmonary side of the ductus before the embolisation coils were put in place. Both dogs remained healthy during a follow-up period of nine months.

PATENT ductus arteriosus (PDA) is the most common congenital cardiovascular anomaly in dogs (Ackerman and others 1978). Medical treatment with a prostaglandin antagonist has been described in one dog by Atwell (1977), but surgical closure has been the treatment of choice for many years (Birchard and others 1990). Recently, catheterisation techniques have been described (Snaps and others 1995, Grifka and others 1996, Schneider and others 1996, Fellows and others 1998, Fox and others 1998), which have the advantages over surgery that they do not require a thoracotomy, need minimal postoperative care and have a shorter recovery period.

Closure with spring coils is the usual procedure for small PDAs, but these coils are considered unsuitable for PDAs which do not have a focal narrowing at the pulmonary ostium or which are larger than 5 mm (Sharafuddin and others 1996, Schneider and others 1998). This paper describes two cases which have been treated successfully by placing a balloon

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