Malformations and the Manx Syndrome in Cats

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SUMMARY

Breeding experiments were conducted on cats with congenital taillessness, to test the dissemination pattern of taillessness in their offspring. Clinical evaluation, radiographic analysis of the vertebral column and histological studies of the digestive tract and central nervous tissue were conducted to determine the association of malformations of these systems in cats born with different degrees of taillessness noted in the rumpy and stumpy cats.

The mode of transmission of the tailless (Manx) condition assumed to be through an autosomal dominant factor (M) was confirmed by this investigation.

It is hypothesized that the problems associated with the tailless condition such as *spina bifida*, urinary and faecal incontinence and locomotor disturbances of the pelvic limbs may all be related to a disturbance affecting the development of the central nervous system in the early embryonic life.

RÉSUMÉ

Les malformations et le syndrome Manx, chez les chats

Cette étude visait à effectuer des expériences de reproduction, chez les chats atteints d'une agénésie caudale, dans le but de déterminer le profil de dissémination de cette anomalie chez leurs descendants. On procéda à une évaluation clinique, à une analyse radiographique de la colonne vertébrale, ainsi qu'à l'étude histologique des systèmes digestif et nerveux central, dans le but de déterminer l'association des malformations de ces systèmes chez les chats nés avec différents degrés d'angénésie caudale, constatés chez les sujets dépourvus de vertèbres coccygiennes et chez ceux dont ces vertèbres présentaient des anomalies.

Cette étude permit de confirmer les soupçons selon lesquels cette agénésie caudale serait attribuable à un gène autosome dominant (M), chez les chats Manx.

On suppose que le *spina-bifida*, l'incontinence fécale et urinaire et les troubles locomoteurs des membres pelviens, autant de problèmes reliés à l'agénésie caudale, pourraient tous résulter d'un trouble qui affecte le développement normal du système nerveux central.

INTRODUCTION

Congenital defects are defined as abnormalities of structure or function present at birth, and may effect many or perhaps all structures of the body. Many different congenital defects have been identified in man and their counterparts are frequently seen in domestic animals. Every body system is susceptible to congenital malformations, but the nervous system is one of the most commonly affected in both domestic animals and man. A majority of these malformations can be recognized by structural changes of the nervous system alone, or by changes in both the axial skeleton and the central nervous system.

Cats regarded as tailless, although often with a very short tail, are worldwide in distribution. The occurrence of tailless cats in the relatively isolated area known as the Isle of Man in the Irish Sea has attracted considerable attention for over a century.

Data presented by several researchers (4, 15, 16) indicate that Manx cats are actually not all tailless, but are subject to all degrees of taillessness. Litters resulting from Manx to Manx matings contain kittens which have no coccygeal vertebrae (rumpy), several (1-7) coccygeal vertebrae fused in an upright position (rumpy-riser), and several (2-14) coccygeal vertebrae which may have a severe kink resulting from abnormal vertebrae (stumpy) and

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normal tails (4). The distinction between rumpy-riser and stumpy is based on the ability of the latter to move the coccygeal vertebrae laterally, this movement being impossible in rumpy-risers (16). Within each of the first three aforementioned categories there is considerable variability, but in all known cases, the vertebrae are abnormal.

The tailless appearance of the Manx cat constitutes the relatively normal end of a spectrum of genetically controlled breed characteristics which include several serious deficiencies and potentially lethal abnormalities (6). Despite their unusual appearance, many Manx cats are successful show animals or valued pets without signs of any other severe disorders. However, the Manx breeder soon discovers that a considerable percentage of kittens suffer from severe congenital abnormalities primarly related to spinal cord lesions (6). Breeding experiments were conducted in our laboratory on "tailless" cats believed to have originated on the Isle of Man, in order to test the inheritance of the different types of tailless. Clinical evaluations, radiographic analyses of the vertebral column, and histological examinations of the digestive tract and central nervous system were carried out on the litters obtained in various breeding experiments and on cats donated for study by various breeders to study the congenital malformations frequently associated with the Manx trait.

MATERIALS AND METHODS

Animals

The animals used in this study were the tailless (Manx) cats donated by breeders from Canada and the United States. The cats used for breeding purposes were all "healthy" Manx cats. Histological studies were also extended to clinically abnormal animals presented for euthanasia and pathological examination.

Animals were classified in terms of the Manx trait on the basis of the number of sacral and coccygeal vertebrae present (6) and mobility of the tail (16). The number of vertebrae present was determined from radiographs taken with the animals lying in lateral recumbency, using a Phillips 300 Ma fullwave rectifying x-ray machine with Kodak X-R I film

Histological Examination

Animals were euthanized with Euthanyl Forte Solution[1], following which pieces of tissue were fixed in 10% neutral buffered formalin for 48 hours and were rinsed in water for four hours prior to dehydration and paraffin embedding. Sections 5-6 μm in thickness were cut on an American Optical Rotary Microtome, and were stained with haematoxylin and eosin and mounted in DPX. Slides were examined and photographs were taken on a Zeiss Photomicroscope III with Panatomic-X.

RESULTS

Radiographic Findings and Clinical Evaluation

Breeding Colony Animals — All animals used for breeding purposes were clinically normal in terms of bowel and bladder function and locomotor ability. Lateral spinal radiographs of the individual animals used in the breeding experiments enabled the classification of these cats in terms of tail length and configuration (Figure 1). Animals were designated as rumpy, rumpy-riser, stumpy and longtail using the system of classification described elsewhere (6). The system of classification was expanded to include animals with up to 15 coccygeal vertebrae as stumpies. Radiographic findings are summarized in Table I.

Of the two males used for breeding, one was a stumpy and the other was a rumpy. The radiographic features of the rumpy male (Warpaint) were not available as he was not a resident of the breeding colony. The litter he sired was conceived elsewhere and the pregnant female cat (Chantilly) was sent to this laboratory. Warpaint was used only once in the present study, whereas the stumpy male (Red) was mated several times with females of various tail characteristics. One of the females (Flower Power) was erroneously identified as a stumpy prior to inclusion in the breeding program. The correct identification as a docked longtail was determined at a later date.

[1]Euthanyl Forte Solution, MTC, Hamilton, Canada.

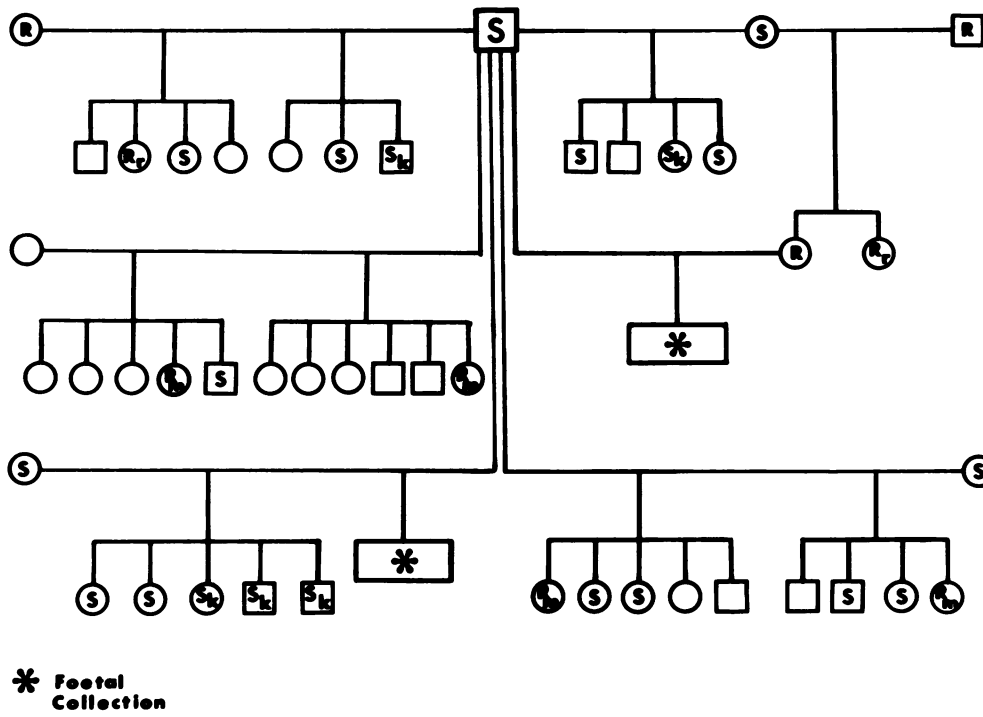

FIGURE 1. Pedigree chart of the Manx breeding colony. R: rumpy, Rr: rumpy-riser, S: stumpy, Sk: stumpy with tail kinks, Rm: rumpy with megacolon.
*Foetal collection by Caesarean section.

The normal cat has seven cervical, 12 thoracic, seven lumbar, three sacral and 18-23 coccygeal vertebrae. The number of sacral vertebrae (three) was similar in the stumpy, rumpy and longtail cats used for breeding. The most consistent number of lumbar vertebrae noted was seven, with one exception (Elsa) which had only five. Coccygeal vertebrae were absent in rumpies and ranged from five to seven in the stumpies (Table I).

Kittens born in the breeding colony during the course of this study were radiographed and classified in terms of tail characteristics. In all, ten litters were obtained from matings involving at least one parent with a tail malformation. Kittens of each of these litters were examined

TABLE I

VERTEBRAL DESCRIPTION AND RADIOGRAPHIC FINDINGS OF THE MANX CATS USED FOR BREEDING

Identification			Number of Vertebrae			Classification
Number	Name	Sex	Lumbar	Sacral	Coccygeal	
1	Chantilly	F	7	3	5	Stumpy
2	Flower Power	F	7	3	4[a]	Longtail
3	Fanny	F	7	3	5	Stumpy
4	Elsa	F	5	3	6	Stumpy
5	Pandora	F	7	3	0	Rumpy
6	Silver	F	7	3	0	Rumpy
7	Red	M	7	3	7	Stumpy
8	Warpaint	M	— Unknown —			Rumpy

[a] Docked longtail

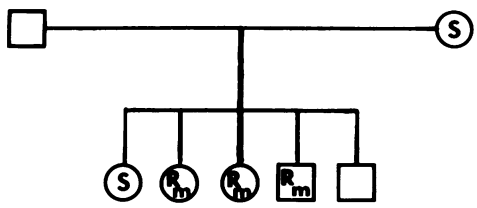

FIGURE 2. Pedigree chart of a Manx family unrelated to the original breeding colony shown in Figure 1.

for the presence of clinical problems and vertebral anomalies. Nine of these litters were born within the colony (Figure 1). One litter, which was born elsewhere, was donated for study (Figure 2).

The classification of the kittens in terms of radiographic tail characteristics and clinical evaluation is summarized in Tables II to IV.

Three litters were obtained from rumpy-stumpy matings (Table II). One rumpy (with no coccygeal vertebrae) and two rumpy-risers (with one coccygeal vertebra) were noted among the kittens obtained in matings in which one of the parents was a rumpy. Both kittens in the litter sired by a rumpy (Warpaint) showed taillessness, one being a rumpy, the other being a rumpy-riser. Stumpies and longtails were obtained both times when the stumpy male (Red) was mated to a rumpy female (Pandora). In one of the rumpy kittens from a litter of Pandora and Red, a tail kink involving the ninth and tenth coccygeal vertebrae was noted. All nine kittens were normal with regard to bowel and bladder function and locomotor ability (Table II).

Four litters were obtained in stumpy-stumpy matings (Table III). In one litter (from Fanny and Red) all kittens were clinically normal stumpies with four out of five showing tail kinks in various regions. The two litters obtained from Elsa and Red consisted of rumpy, stumpy and longtail kittens. The rumpies of these litters had megacolon and, in addition, one of them exhibited urinary incontinence and paresis of the pelvic limbs. The litter from Chantilly and Red consisted of one longtail and three stumpies, including one with a tail kink. All of these kittens were clinically normal.

In the three litters obtained by mating stumpy Manx to long tailed cats, three classes of kittens (rumpy, stumpy and longtail) were noted (Table IV). In each of the two matings of Flower Power to Red, a rumpy kitten exhibiting not only the absence of coccygeal vertebrae but also a reduction of sacral vertebrae was noted. In one of these kittens, a reduced number of lumbar vertebrae was also noted, along with a congenital anogenital malformation (Figure 3). These rumpies also exhibited signs of megacolon.

The litter from a stumpy female and a longtail male consisted of three rumpies, a stumpy and a longtail. The rumpies had a reduced number of sacral vertebrae and one of them also had a reduced number of lumbar vertebrae. All of these rumpies had megacolon and one of them exhibited paraparesis and urinary incontinence.

The longtailed kittens from stumpy-longtail matings (Table IV) and stumpy-rumpy and stumpy-stumpy matings (Tab-

TABLE II
LITTER DESCRIPTION AND VERTEBRAL COLUMN FEATURES OF THE KITTENS OBTAINED BY MATING RUMPY WITH STUMPY MANX CATS

Dam	Sire	Number of Kittens	Sex	Number of Vertebrae			Classification	Clinical Evaluation
				Lumbar	Sacral	Coccygeal		
Chantillyx (Stumpy)	Warpaint (Rumpy)	2	F	7	3	0	Rumpy	Normal
			F	7	3	1	Rumpy-riser	Normal
Pandora x (Rumpy)	Red (Stumpy)	4	F	7	3	1	Rumpy-riser	Normal
			F	7	3	7	Stumpy	Normal
			M	7	3	23	Longtail	Normal
			F	7	3	22	Longtail	Normal
Pandora x (Rumpy)	Red (Stumpy)	3	F	7	3	22	Longtail	Normal
			F	7	3	9	Stumpy	Normal
			M	7	3	15;K 9-10	Stumpy	Normal

K: tail kink (followed by numbers which indicate the vertebrae involved in the kink)

TABLE III
LITTER DESCRIPTION AND VERTEBRAL COLUMN FEATURES OF THE KITTENS OBTAINED BY INTERBREEDING STUMPY MANX CATS

Dam	Sire	No. of Kittens	Sex	Number of Vertebrae			Classification	Clinical Evaluation
				Lumbar	Sacral	Coccygeal		
Fanny	Red	5	F	7	3	7;K 3-4, 5-6	Stumpy	Normal
(Stumpy)	(Stumpy)		F	7	3	3	Stumpy	Normal
			F	7	3	14;K 7-8	Stumpy	Normal
			M	7	3	15;K 6-7, 13-15	Stumpy	Normal
			M	7	3	15;K 2-4, 6-8, 12-13, 14-15		
Elsa	Red	4	F	7	3	8	Stumpy	Normal
(Stumpy)	(Stumpy)		F	6	2	0	Rumpy	Megacolon
			M	7	3	9	Stumpy	Normal
			M	7	3	19	Longtail	Normal
Elsa	Red	5	F	7	3	6	Stumpy	Normal
(Stumpy)	(Stumpy)		F	7	3	6	Stumpy	Normal
			F	6	2	0	Rumpy	Paraparesis, urinary incontinence, megacolon
			F	7	3	22	Longtail	Normal
			M	7	3	23	Longtail	Normal
Chantilly	Red	4	F	7	3	7	Stumpy	Normal
(Stumpy)	(Stumpy)		F	7	3	13;K 6-7	Stumpy	Normal
			M	7	3	5	Stumpy	Normal
			M	7	3	20	Longtail	Normal

K: tail kink (numbers indicate the vertebrae involved in the kink).

TABLE IV
LITTER DESCRIPTION AND VERTEBRAL COLUMN FEATURES OF THE KITTENS OBTAINED BY MATING STUMPY MANX CATS WITH LONGTAIL CATS

Dam	Sire	No. of Kittens	Sex	Number of Vertebrae			Classification	Clinical Evaluation
				Lumbar	Sacral	Coccygeal		
Flower Power (Longtail)	x Red (Stumpy)	5	F	6	2	0	Rumpy	Megacolon, anogenital malformation
			F	7	3	23	Longtail	Normal
			M	7	2	6	Stumpy	Normal
			M	7	3	21	Longtail	Normal
			M	7	3	23	Longtail	Normal
Flower Power (longtail)	x Red (Stumpy)	6	F	7	2	0	Rumpy	Megacolon
			F	7	3	23	Longtail	Normal
			F	7	3	23	Longtail	Normal
			M	7	3	22	Longtail	Normal
			M	7	3	21	Longtail	Normal
			M	7	3	23	Longtail	Normal
Stumpy	x Unknown (Longtail)	5	F	7	3	11	Stumpy	Normal
			M	6	1	0	Rumpy	Megacolon
			F	7	2	0	Rumpy	Paraparesis, urinary incontinence, megacolon
			F	7	1	0	Rumpy	Megacolon
			M	7	3	23	Longtail	Normal

FIGURE 3. Ventral aspect of a female rumpy kitten from a longtail (Flower Power) and stumpy (Red) mating showing a single anogenital orifice.

les II and III) consistently exhibited seven lumbar and three sacral vertebrae and from 19 to 23 clinical problems.

Manx Cats Donated by Breeders — The Manx cats donated for study were subjected to radiographic and clinical evaluations and the findings are summarized in Tables V and VI. Myelographic examination of these cats (Figure 4) allowed identification of two cases of *spina bifida occulta*

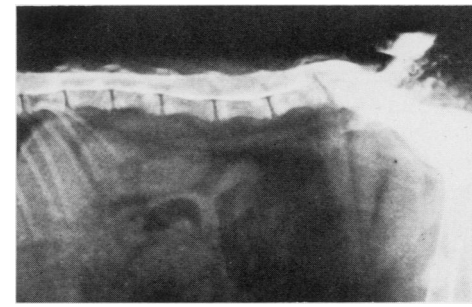

FIGURE 4. Myelogram of female rumpy kitten (Punkin) showing evidence of a myelocoele. Note pooling of contrast media subcutaneously in lumbosacral region.

(Punkin and Danny). Both animals were born with a meningocele located at the base of the spine. This had subsequently healed, but as the kittens grew older, they showed signs of urinary and faecal incontinence and locomotor problems involving the hindlimbs (Figure 5). The principal gait problem consisted of a plantigrade posture when walking or standing. Rapid locomotion in these animals was possible only by hopping. In addition, both animals had reduced cutaneous sensation in the perineal region and one animal (Punkin) had a mild rectal prolapse (Figure 6). The myelograms of these animals (Figure 4) showed attachment of the spinal cord to the subcutaneous tissues in the lumbar region.

Gross Pathological Findings

Postmortem examinations of the Manx

TABLE V

RADIOGRAPHIC FINDINGS AND CLASSIFICATION OF MANX CATS DONATED FOR STUDY BY BREEDERS

Identification			Number of Vertebrae			
Number	Name	Sex	Lumbar	Sacral	Coccygeal	Classification
9	Danny	M	7	2	0	Rumpy
10	Giarey	F	6	1	0	Rumpy
11	Downey	F	7	3	5	Stumpy
12	Punkin[a]	F	7	2	0	Rumpy
13	Blue-Cream[a]	F	7	3	2	Rumpy-riser
14	Davies	F	7	2	0	Rumpy
15	B.J.	F	6	1	0	Rumpy
16	Bryanne	F	7	0	0	Rumpy
17	Fukyu	F	7	2	0	Rumpy
18	Cloudy	M	7	3	0	Rumpy

[a] Littermates

cats with clinical abnormalities and of four normal cats revealed signs of faecal incontinence in the abnormal animals. The signs included faecal staining of the perineum and abdominal distension in the affected cats. Similar necropsy findings in all cases of faecal incontinence, included marked enlargement of the colon. The distended segment extended from the rectum to the caecum in most cases (Figure 7). Upon opening the distended segment of bowel, hard dry fecoliths were observed, proximal to which small amounts of loose fecal material were present. Six of the cats also had urinary incontinence (Table VI). Greatly enlarged urinary bladders were observed in five of these animals (Figure 8). In one animal, a mature male (Danny), the bladder appeared to be shrunken, firm and inflamed.

Grossly visible spinal cord malformations were observed in all Manx cats with clinical abnormalities. In one of the Manx cats (Punkin) which had urinary and faecal incontinence and hindlimb incoordination, the spinal cord appeared normal in the anterior region, but just beyond the caudal border of the sacrum it terminated by joining the dura which was covered by subcutaneous fat and skin. This had been established previously by myelography (Figure 4). Traction of the fur of this animal in this region pulled down the lower spinal cord. Much of the sacral cord

TABLE VI

DESCRIPTION AND CLINICAL EVALUATION OF ABNORMAL ANIMALS SENT FOR STUDY

Identification					
Number	Name	Sex	Category	Age	Clinical Signs
9	Danny	Male	Rumpy	3y	Monoplegic, with urinary incontinence and chronic megacolon, and reduced cutaneous sensation in perineal region. Failed to show any interest in breeding queens in estrus
10	Giarey	Female	Rumpy	3m	Paraparesis, with gait progression by hopping; urinary incontinence; megacolon and reduced cutaneous sensation in the perineal region
11	Downey	Female	Stumpy	4m	Megacolon
12	Punkin[a]	Female	Rumpy	3m	Paraparesis, with gait progression by hopping; urinary incontinence; megacolon
13	Blue[a]	Female	Rumpy-riser	3m	Megacolon
14	Davies	Female	Rumpy	3m	Megacolon
15	B.J.	Female	Rumpy	5w	Megacolon
16	Bryanne	Female	Rumpy	10w	Paraparesis; megacolon
17	Fukyu	Female	Rumpy	2m	Paraparesis; urinary incontinence; megacolon
18	Cloudy	Male	Rumpy	4m	Megacolon

y = years, m = months, w = weeks

[a] Littermates

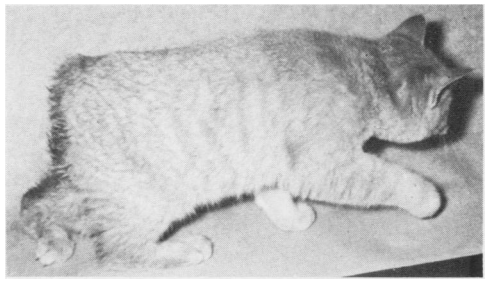

FIGURE 5. Rumpy female kitten (Punkin) from rumpy-rumpy mating. Note the plantigrade posture and medial rotation of the hindlimbs.

was absent and no coccygeal vertebrae were present.

Similar abnormalities were observed in a male Manx cat (Danny). This animal, which was three years of age at the time of euthanasia, had also failed to mate with any female during a period of almost one year when he was part of the breeding colony. In all cats with clinical abnormalities comparable spinal cord malformations were observed. In most cases, these involved partial or complete absence of the sacral and caudal segments of the spinal cord, in conjunction with sacral agenesis or dysgenesis.

All cats with clinical problems were rumpies with the exception of one cat

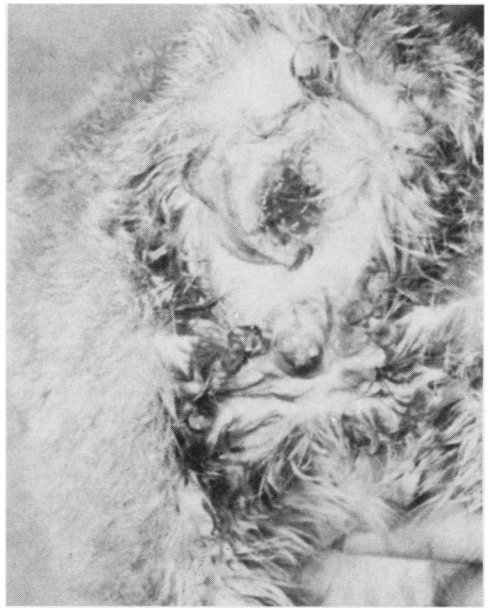

FIGURE 6. Perineal region of kitten in Figure 5 showing partial rectal prolapse. Staining of the hair coat in this region can be attributed to faecal and urinary incontinence. In addition, this kitten had *spina bifida occulta*.

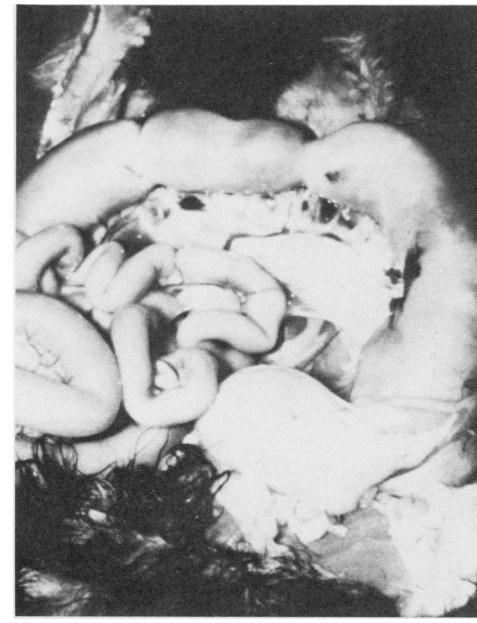

FIGURE 7. Exposed viscera of rumpy female kitten showing great distention of the large intestine (megacolon).

(Downey) which was a stumpy. This cat had shown signs of faecal incontinence. In this animal the vertebral arches of the caudal lumbar and sacral vertebrae were bifid, connected only by a thin dorsal membrane. The spinal cord in this region had a dorsally located slit-like cavitation covered by intact meninges.

FIGURE 8. Viceral view of the above kitten showing highly distended bladder. This kitten had both urinary and faecal incontinence.

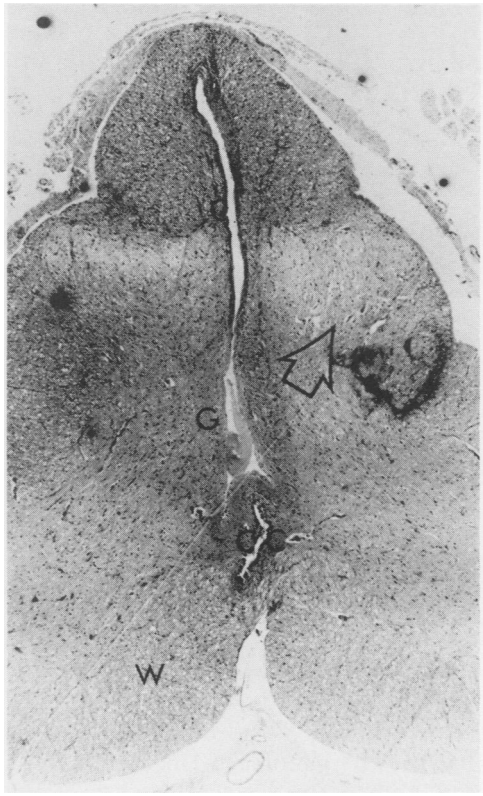

FIGURE 9. Section of spinal cord from rumpy Manx (Punkin) with gait problems involving the hindlimbs. Note the presence of a cavity (C) indicating syringomylia within the dorsal columns of the spinal cord, with surrounding degeneration involving dorsomedial columns (arrow). CC: central canal, W: white matter, G: grey matter. H & E. X25.

Histopathological Findings

Histological examination of the abdominal and thoracic cavity organs and central nervous tissue revealed that the most significant abnormalities were in the colon and caudal spinal cord. Paraffin and semithin sections of colon showed evidence of hyaline degeneration of the smooth muscle layers and degeneration of ganglion cells in Auerbach's and Meissner's plexus was evident. The numbers of ganglion cells in the Auerbach's plexus of the colon were comparable in animals with megacolon and normal cats. However, the appearance of the ganglion cells was abnormal in that they contained many vacuoles and were undergoing chromatolysis. Macrophages were present in many sections. Other areas of the gastrointestinal tract of cats with megacolon were apparently normal.

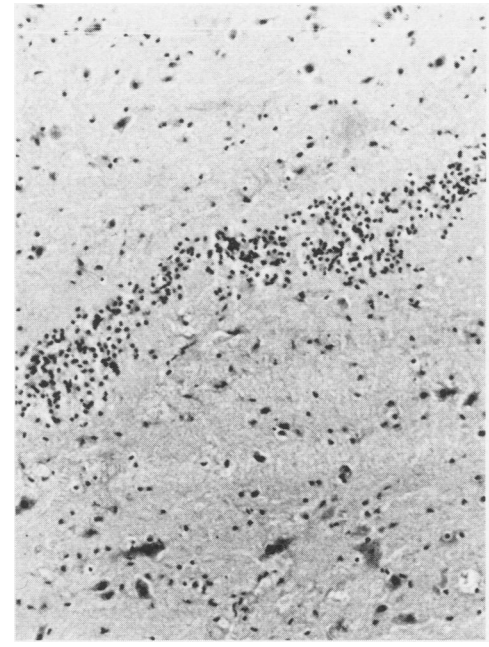

FIGURE 10. Section of lumbar spinal cord from female rumpy-riser (Blue Cream) with megacolon. Note the aggregate of astrocytes. This was the only abnormality which was microscopically evident in this animal. H & E. X160.

Examination of sections of spinal cord revealed that the cervical, thoracic and upper lumbar regions were generally normal. However, in animals with incontinence of both urine and faeces and with gait problems in the hindlimbs, the spinal cord in each case showed cavitation of the dorsal white matter with surrounding tract degeneration which was most severe in the dorsomedial columns (Figure 9). The cavitation was apparently continuous, extending from the middle of the lumbar region to the lowest sacral segment. Its position in all sections suggested the presence of one continuous ramifying cavity which communicated in some sections with the central canal of the spinal cord. The cavity contained an eosinophilic amorphous material, probably cerebrospinal in origin (Figure 9). Demyelinated axons and numerous astrocytes surrounded the cavity.

In animals with only megacolon, less significant histopathological features were noted in the spinal cord. In these, aggregates of astrocytes in the lumbar and sacral cord segments (Figure 10) were the most consistent change observed microscopically.

DISCUSSION

Many of the cats examined during the course of the present study showed evidence of clinical problems and developmental defects, all of which are believed to be associated with the most conspicuous congenital malformation, taillessness. Although taillessness is the most easily observable alteration in the Manx cat, postmortem examinations conducted during this and previous studies indicate that the spinal cord is highly abnormal in animals with problems such as *spina bifida*, incontinence of faeces and urine, perineal sensory loss and abnormal hindlimb action (5, 6, 9). In these cases, the spinal cord ended prematurely with an absence of certain spinal nerves from the sacral cord segments which supply innervation to the colon, bladder, hindlimbs and perineal region (12). The peculiar hopping gait observed in Manx cats with syringomyelia observed in this study and by others (8, 9) is especially noteworthy. Similar findings were observed in a breed of dog with comparable spinal cord lesions (10).

It would appear that, besides morphological variation in the coccygeal and sacral vertebrae, there are movement abnormalities, incontinence and neuroschisis in Manx cats, all of which may have been produced by the action of the gene that adversely affects the development of the neural tube (8).

The absence of part of the spinal cord in Manx cats is highly significant to the genesis of taillessness. The embryonic neural tube, together with the notochord, has been shown to be involved in the morphogenesis of somite-derived vetebral cartilage in the chick embryo *in vivo* (17) and *in vitro* (2). Similar tissue interactions are believed to occur in vertebral column development in mammals. Hence, it can be postulated that an underlying defect of the developing central nervous system may result in aberrant development of the caudal vertebral column in Manx cats. It can be further hypothesized that this defect might involve partial agenesis of dysgenesis of the neural tube, with subsequent failure to release some messenger substance essential for early somite chondrogenesis (11). This theory appears to be very plausible in the light of the generalized malformations of the neural tube in supposedly homozygous Manx embryos. It is postulated that the effects of the Manx gene (M) are more localized in the heterozygote Manx cat, affecting only the caudal aspects of the neural tube and overlying vertebrae.

The neural tube defects observed in the abnormal Manx foeti (3) bear special importance in predicting the pathogenesis of *spina bifida* observed in some Manx cats. The nervous system is derived from a thickened plate of ectoderm located in the middorsal line of the early embryo (1). As the lateral parts of this plate grow dorsal and become elevated, a neural groove is formed. This can be recognized in cat embryos of four somites (14). These elevated folds approach one another dorsally in the midline, forming the neural tube. Closure begins at the junction of the future spinal cord and brain and progresses both cephalad and caudad. Closure of the neural tube is complete in the cat embryo of 16 somites (14). In normal human embryos, closure is completed rostrally at the 18-20 somite stage and caudally somewhat later, in embryos of approximately 25 somites (18). With the caudal closure of the neural tube, the primitive central nervous system is established. Thus, *spina bifida* or *cranium bifidum*, which follow incomplete closure of the neural tube, are determined in very early embryonic life.

There are two main theories concerning the basic embryogenesis of *spina bifida*. Some investigators believe that neurulation occurs and a subsequent rupture of the hydromyelic spinal cord ultimately results in the reformation of a neural plate (18). Others believe that neurulation is not completed and therefore the neural tube does not develop, leaving an open neural plate (7, 13, 19). The second theory has generally received much wider acceptance (18). In cases of anencephaly in man, rostral interference with neural tube closure is believed to be related to faulty folding and overgrowth of brain tissue with failure of development of dorsal meninges and cranial bones (18). A similar situation may occur in the caudal end of the neural plate where there is overgrowth of neural tissue, failure of development of dorsal meninges and/or neural arches. Spinal

nerves may be stretched or torn by abnormal movements of the neural plate, or may be absent altogether (18). The clinical signs of faecal and urinary incontinence, perianal sensory loss and lower limb paralysis or paresis observed in humans with *spina bifida* and Manx cats may all be causally related to the abnormal movement of the neural plate during the development of the embryo that carries the Manx gene.

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