

DERMOMYOSITIS OF ASCARIS LUMBRICOIDES (NEMATODA:ASCARIDEA)
IN CANADA

A THESIS
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ABSTRACT

Dermomyositis of Ascaris lumbricoides was found for the first time in Canada (Winnipeg, Manitoba, Vancouver, British Columbia and Montreal, Quebec). Its average incidence in the 14,441 ascarids examined was 1.9 per cent. It occurs more frequently in the autumn, 2.2 per cent, than in the spring, 1.5 per cent, and more often in females, 2.1 per cent, than in males, 1.3 per cent.

The histological examination of the lesions revealed a hitherto unknown phenomenon, the vacuolation of both the hypertrophied hypodermis and of the adjacent muscles.

Two types of fungi and one of bacteria were found in the dermomyositic lesions. This is the first finding of fungi in such lesions.

ACKNOWLEDGEMENTS

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I. HISTORICAL REVIEW AND STATEMENT OF THE PROBLEM

In 1912 Weinberg and Keilin (39) described cuticular lesions in Ascaris megalcephala Cloquet, 1824 in Paris, and named the disease "dermomyositis". The lesions are hard, brown or yellow plaques, with their longitudinal axis at right angles to that of the worm, or in other cases, as longitudinal striations irregularly distributed over the body surface. Cleland and Johnston (9), also in 1912, described small crateriform projections, about the size of a pin's head scattered over the cuticle of Ascaris suilla Dujardin, 1845 from pigs in Sydney, Australia. Manter (28) in 1929 found lesions of the cuticle in ascarids from hogs in Nebraska, but did not identify them with the disease described by Weinberg and Keilin or Cleland and Johnston. In 1931 Lubinsky (24) working in Kiev, Ukraine found dermomyositis in Ascaris lumbricoides L, 1758 from man and pigs (referring to pig ascarids as A. suilla), and also in Ascaris equorum Goeze, 1782, Oxyuris curvula Rudolphi, 1803, Strongylus equinus Mueller, 1780 and S. vulgaris (Looss, 1900) from horses, Toxascaris limbata Railliet and Henry, 1911 from dogs and Heterakis perspicillum (Rudolphi, 1803) from chickens. His illustrations of dermomyositis of A. lumbricoides were reproduced by Dollfus (11). In a subsequent paper Lubinsky (25) described the histopathology of dermomyositis in the pig ascarid. Pavlovskii (32) in 1934 in Leningrad, Russia reported that pigmented

spots on the cuticle of ascarids were produced by bacteria (Bacterium ascaridianum). Stewart and Godwin (34,35) in 1959 and in 1963 found dermomyositis in pig ascarids from Tifton, Georgia and studied the etiology and histopathology of this disease. They isolated a species of Pseudomonas, a rod shaped bacterium, from cuticular lesions of ascarids and regard this micro-organism as the cause of the disease. In using this strain of Pseudomonas they experimentally produced lesions in A. lumbricoides both in vitro and in vivo.

In the present paper an attempt is made to study the following:

1. The geographical distribution and incidence of dermomyositis of pig ascarids in Canada.
2. The seasonal variation of its occurrence.
3. Sex differences in occurrence.
4. The histopathology of dermomyositis.

II. MATERIALS AND METHODS

Specimens of Ascaris lumbricoides were obtained from pigs at two Winnipeg abattoirs, Canada Packers Ltd., and Swift Canadian Ltd. Material fixed in 10% formalin was also received from Mr. P. T. K. Woo, University of British Columbia, Vancouver, and from Dr. R. P. Harpur, McGill University, Montreal. Live specimens were transported to the laboratory in a pail and washed in warm water to remove any debris. The live worms were examined with a hand lens for dermomyositic lesions, sexed and counted. Ascarids from Mr. Woo had already been examined by him and only those with lesions (17) were received. The worms (2,019) collected by Dr. Harpur were shipped in bulk and examined here. Specimens for macrophotography were fixed in 10% formalin. A total of 14,441 ascarids were examined for this project.

METHOD OF COUNTING LESIONS.

Each worm with numerous lesions (150 or more) was divided into four quarters. Two cylindrical portions 5 mm. long were cut at random from each quarter. Each sample then was slit lengthwise, the internal organs removed, and the remaining integument placed between two slides. The lesions were counted using a dissecting microscope. The number of lesions per unit area based on the examination of these samples was recorded for each quarter of the worm.

The surface area of each worm was determined by regarding the anterior and posterior three centimetres as cones and the remainder of the worm as a cylinder. The number of lesions per square centimetre of body surface for each of the 46 worms was calculated and recorded. (See appendix A).

The lesions of worms with 20 or less were counted directly.

HISTOLOGICAL METHODS.

Four to five mm. long portions of worms with lesions were fixed in 4% formaldehyde or Formalin-Alcohol-Acetic (FAA) for 24 hours, dehydrated in ethyl alcohol, cleared in xylene, embedded in Tissuemat M.P. 61° C. (Canlab Supplies Ltd.), and sectioned at 8 to 10 μ .

Two points are worthy of mention: it was necessary to ensure that the microtome blade struck the scab portion of the block first and thus when cutting did not tear it from the soft tissues. In some sections the cuticle tended to shrink and to become detached from the slide. This was more pronounced when working with formalin fixed material than with that fixed in FAA. Sections were stained with acid fuchsin and Mallory's counterstain (27), Heidenhain's iron haematoxylin (27), and Ehrlich's haematoxylin and eosin. Acid fuchsin and Mallory's.

From 50% alcohol, sections were transferred into acid fuchsin for about 30 seconds, rinsed in water, stained with

Mallory's counterstain for 5-7 minutes, rinsed in water again and differentiated in 95% alcohol until the colours became clean and vivid.

Heidenhain's iron haematoxylin.

Sections were mordanted in 4% iron ammonium alum for 2 hours, stained with Heidenhain's haematoxylin for 2 hours, differentiated in 2% mordant under a microscope and blued in running water for 30 minutes.

Haematoxylin and eosin.

Sections were stained with Ehrlich's haematoxylin for 5-10 minutes, rinsed in water, differentiated with acid alcohol (75% alcohol and 0.5% conc. HCL) for a few seconds, then blued in running water for 30 minutes and counter-stained with eosin.

All slides were dehydrated in absolute alcohol, cleared in xylene and mounted in Permount.

MACROPHOTOGRAPHY.

A Zeiss 4" X 5" Universal camera and a Nikon 35 mm. camera were used. Specimens were placed in a tray made of an 8" X 16" X 1" frame of wood mounted on a sheet of glass with the aid of aquarium cement. The tray was placed on top of two 6" high blocks of wood on black cardboard as a background. The worm, or portion thereof, was placed on the tray and covered with a small sheet of glass supported by glass rods. The space between the glass sheets was slowly filled with distilled water and any bubbles carefully removed. The whole apparatus was placed under the camera and illuminated by two photoflood lamps.

MICROPHOTOGRAPHY.

A Zeiss photomicroscope with planochromatic objectives was used. Black and white photographs were made with Agfa IFF film (ASA 25). Kodak Ektachrome-X (ASA 64) and Kodacolor-X (ASA 80) were used for colour photographs.

III. OBSERVATIONS

1. OCCURRENCE AND DISTRIBUTION OF DERMOMYOSITIS IN CANADA.

I attempted to determine the incidence of dermomyositis, according to geographical region, season, and sex of the parasite. The number of ascarids examined, the number affected and the numerical relations between sexes are summarized in Tables I - III.

Eleven thousand six hundred and fifty seven ascarids from Manitoba were examined, and 226, thus 1.9 ± 0.1 per cent*, found with lesions. The corresponding figures for British Columbia were 765 ascarids examined, 17 with lesions or 2.2 ± 0.5 per cent, and for the province of Quebec 2019 worms of which 26 were affected or 1.3 ± 0.2 per cent.

In Winnipeg, where the material from Manitoba was collected, the percentage of ascarids with dermomyositis varied from month to month from 1.3 to 2.5 per cent, the average being 1.9 per cent. Although 500 to 1200 worms were examined monthly, the standard error was high, from 0.3 to 0.7 per cent.

The difference between the incidence of dermomyositis in October, when it was highest, 2.5 ± 0.5 per cent, and March, when it was lowest, 1.3 ± 0.4 per cent, was not significant. Thus the null hypothesis, that the two sampling populations were equal, was accepted. However, even a superficial examination of Table I shows that the figures for the months March to May are considerably lower than those for the months September to November. The

* The standard error of a percentage.

TABLE I

Occurrence of dermatomyositis in Ascaris lumbricoides from pigs.
Material collected in Winnipeg, Man., in 1965.

Month	Number collected			Number with lesions			Percentage with lesions, both sexes
	Total	Female	Male	Total	Female	Male	
Jan.	655	500	155	12	10	2	1.8 [±] 0.5*
Feb.	535	362	173	13	9	4	2.4 [±] 0.7
Mar.	810	514	296	11	8	3	1.3 [±] 0.4
Apr.	1267	845	422	18	16	2	1.4 [±] 0.3
May	945	597	348	18	14	4	1.9 [±] 0.5
June	1037	664	373	16	12	4	1.5 [±] 0.4
July	1050	727	323	17	13	4	1.6 [±] 0.4
Aug.	1061	779	282	27	21	6	2.5 [±] 0.5
Sept.	1146	783	363	26	18	8	2.3 [±] 0.4
Oct.	1093	757	336	27	18	9	2.5 [±] 0.5
Nov.	1026	701	325	20	16	4	1.9 [±] 0.4
Dec.	1032	729	303	21	18	3	2.0 [±] 0.4
TOTAL	11657	7958	3699	226	173	53	1.9 [±] 0.1

* The standard error of a percentage. (12).

TABLE II

Occurrence of dermatomyositis in Ascaris lumbricoides from pigs. Material collected in British Columbia and Quebec, in 1965.

Month	Number collected			Number with lesions			Percentage with lesions, both sexes
	Total	Female	Male	Total	Female	Male	
British Columbia							
June	765			17	10	7	2.2 [±] 0.5
Quebec							
Sept.	863	593	270	8	7	1	0.9 [±] 0.3
Oct.	653	494	159	13	11	2	2.0 [±] 0.5
Nov.	503	355	148	5	5	0	1.0 [±] 0.4
TOTAL	2019	1442	577	26	23	3	1.3 [±] 0.2

TABLE III

Frequency of occurrence of dermatomyositis in male and female Ascaris lumbricoides from Manitoba and Quebec.

Sex	Number of ascarids examined	Number with lesions	Percentage with lesions
Male	4276	56	1.3 [±] 0.2
Female	9400	196	2.1 [±] 0.2

incidence for the months March to May was 1.5 ± 0.2 per cent, that for September to November 2.2 ± 0.3 per cent. The difference, 0.7 per cent, exceeds its standard error, 0.35, two times and is therefore significant at the 0.05 level of the Z-test. To base our groups on still larger numbers of ascarids, we have compared the occurrence of dermatomyositis in the months March to July with the months August to December. January and February were excluded because of the small samples collected, 655 and 535 respectively. In the first period the incidence was 1.6 ± 0.2 per cent and in the second 2.3 ± 0.2 per cent. The difference, 0.7 per cent, exceeds its standard error 2.6 times, and is significant at more than the 0.01 level of probability. There was no significant difference between the occurrence of dermatomyositis in the period December to February (2.1%) and June to August (1.9%). These data, as well as those of Table I, show that the incidence of dermatomyositis is lowest in the spring, increases during the summer, is highest in the autumn, and decreases in the winter months.

It was interesting to study the difference in the occurrence of dermatomyositis between the sexes. Table III, based on material from Manitoba and Quebec, shows that the percentage of males with lesions was 1.3 ± 0.2 , that of females, 2.1 ± 0.2 , the difference being 0.8 per cent. This difference exceeds its standard error 2.85 times, and is significant at at least the 0.01 level of the Z-test.

2. DISTRIBUTION OF LESIONS ON THE BODY SURFACE.

The distribution of lesions on the body surface of 100 affected ascarids was examined in detail. Fifty four of these worms had less than 20 lesions. The frequency of occurrence of various numbers of lesions in this group is represented in Fig.1A. Twenty three out of the 54 ascarids had only 1 lesion, 13 only 2 lesions, and 8 had 3 lesions, thus 44 out of 54 worms had 3 lesions or less (Fig. 1A).

The remaining 46 worms had hundreds or thousands of lesions, the largest number being close to 8000 (See Fig.1B and appendix A). It is interesting that no worms were found to have 20 to 165 lesions.

The number of lesions per quarter of the body length was counted and it was found that the average numbers in the first, second, third and fourth body quarters were 658, 741, 800 and 622 respectively. These figures give the impression that the number of lesions in the anterior and posterior quarters were smaller than in the second and third quarters. One must remember that the middle two quarters are almost cylindrical whereas the oral and caudal ends of the ascarids are conical. Thus the surface of each of the middle quarters is larger than the surface of the anterior and caudal quarters. To decide whether there was "contagious distribution" of lesions it was necessary to find the number of lesions per unit area of body surface in each quarter. In my material the corresponding figures per square centimetre of the four quarters were 91, 83, 88 and 90 lesions. (See appendix A).

The Chi-square test has shown that there was no significant difference between these means. It is obvious that the distribution was random and no particular area was more affected than another.

Fig.1A. The frequency of occurrence of various numbers of lesions in a group of 54 worms.

Fig.1B. The frequency of occurrence of various numbers of lesions in the total sample of 100 worms.

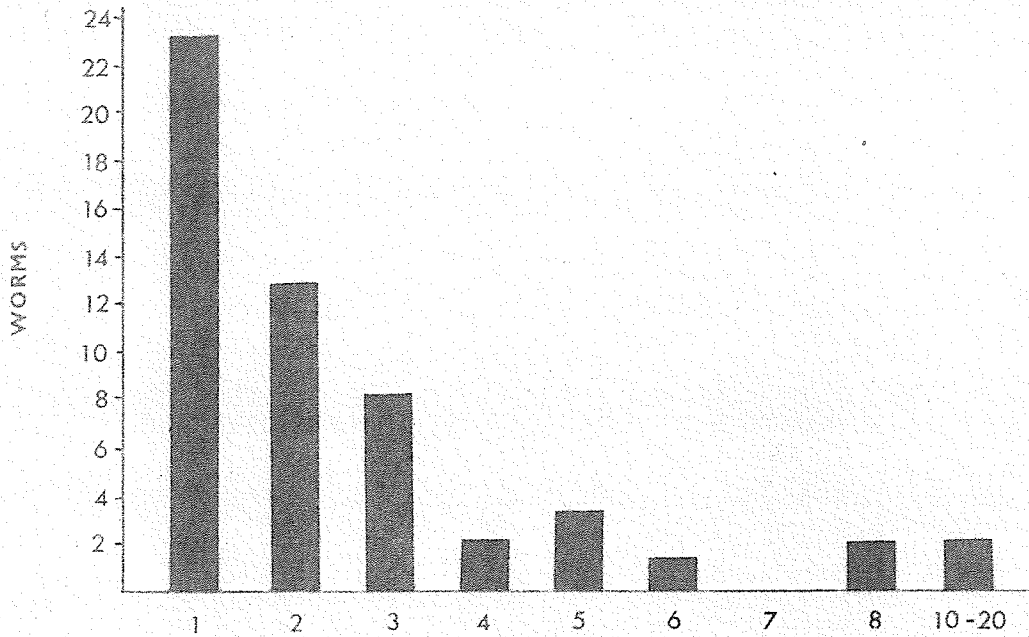


FIG. I.A.

LESIONS

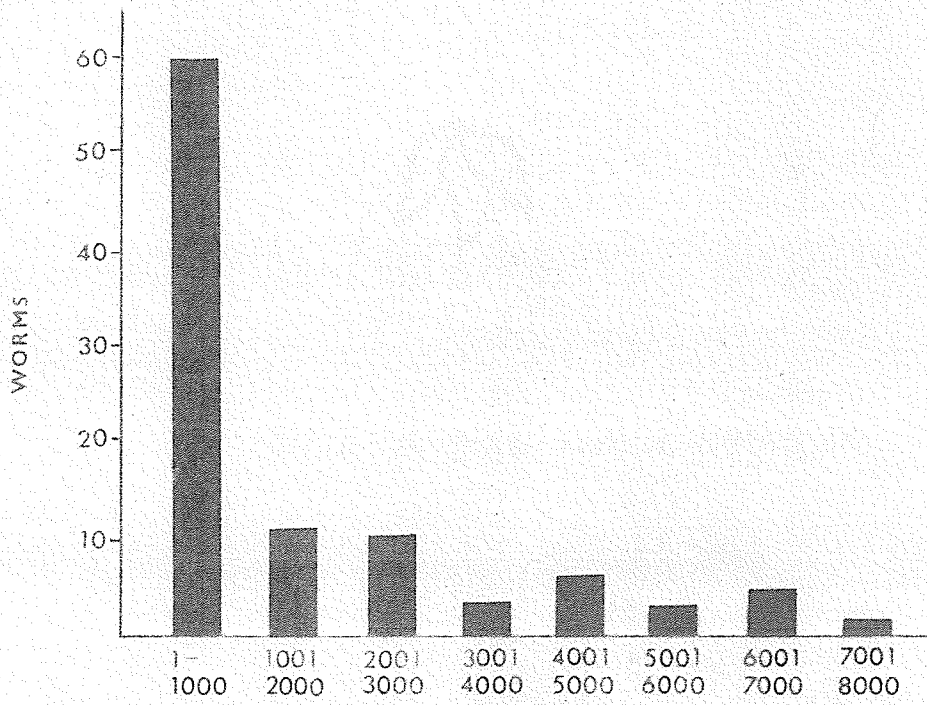


FIG. I.B.

LESIONS

3. MACROSCOPIC APPEARANCE OF DERMOMYOSITIC LESIONS.

Dermomyositis of the pig ascarid manifests itself in lesions of various shapes and sizes (Figs. 2-13, Plates 1-3). The lesions measure 0.5 to 5 mm. and are ellipsoidal or circular, yellow through yellow-green to dark brown in colour. The long axis of these lesions is oriented transversally, thus parallel to the striations of the cuticle. Some lesions look like blisters, their surface being raised and relatively smooth (Fig.2), but in most the surface is rough and cracked (Fig.3). Circular lesions have a light brown raised periphery and a whitish depressed centre (Fig.13). Occasionally the larger lesions are surrounded by a white halo, due to degeneration of the underlying hypodermis and muscles (Fig.12). In many specimens the circular lesions are arranged in longitudinal or oblique rows (Fig.8), in others they are scattered irregularly over the entire surface of the body (Figs.9-10, and 13). In all cases the cuticular striations were obliterated by the lesion.

Many worms had longitudinal rows of dark brown lesions (Figs.4,11). The rows are usually parallel or at a slight angle to the longitudinal axis of the worm with the component lesions of the series being at right angles to this axis. Usually the surface of these lesions is cracked and their edges serrated, perhaps as a result of the movements of the worm (Fig.5).

The largest single lesion found measured about 5 mm. by 2 mm. In the affected region the body was permanently bent away from the lesion, probably because of the extensive damage to the muscles (Fig.12).

The diseased ascarids had all types of dermomyositic lesions described by Lubinsky (24): dermomyositis punctata, linearis, serrata, cruciata and the rupia dermomyositica (Figs.2-13). The smallest, punctiform lesions were the most common, whereas the large ones, of the "rupia" type (Figs.2 and 12) were rare. Of the 46 worms with over 100 lesions, in 33 the "dermomyositis punctata" type and in the remaining 13 the "lineata" type predominated. Although transitional forms between these two types, the coalescence of small lesions into longitudinal rows were common. Transverse expansions of a linear lesion tending to transform it into the "dermomyositis cruciata" were present, but rare (Fig.5). The rare "rupia dermomyositica" occurred only in ascarids with a few (less than 20) lesions (Figs. 2,6,7 and 12). In some "rupias" (Fig.2) the concentric structure of the lesion was well expressed. In many cases the development of the lesions along a scratch of the cuticle was obvious (Figs.4 and 8).

PLATE 1.

Fig. 2. A raised lesion with relatively smooth surface. 40X.

Fig. 3. Small lesion with cracked surface. 40X.

Fig. 4. Longitudinal series of lesions. 30X.

Fig. 5. Serrated edge of longitudinal lesion. 60X.

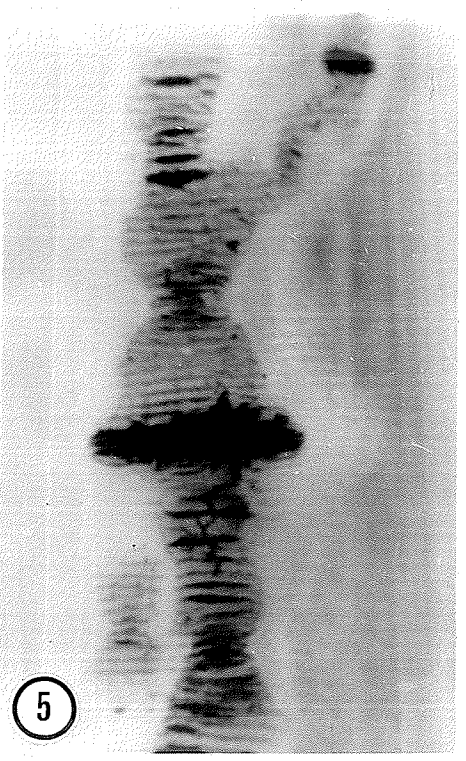
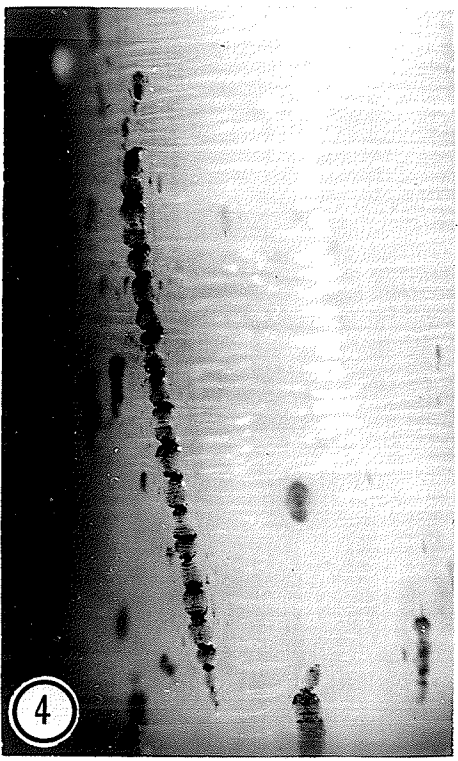
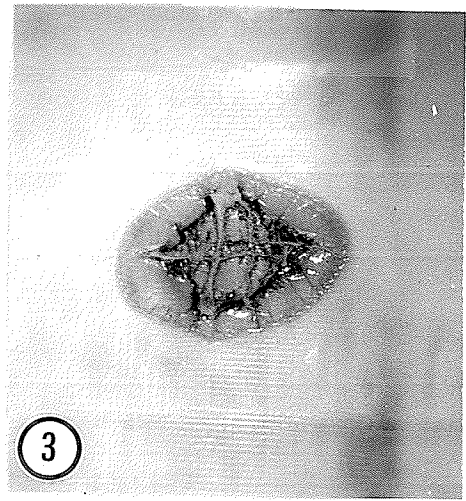
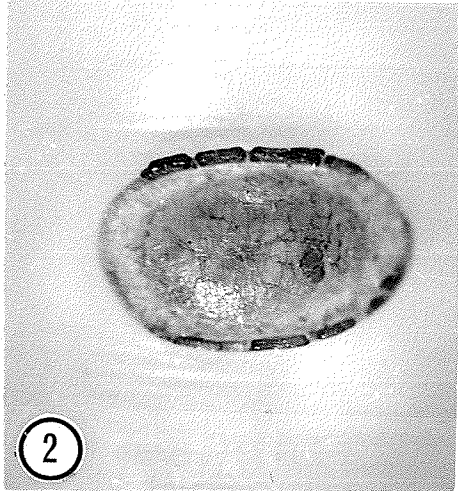


PLATE I

PLATE II.

Figs.6 and 7. Large individual lesion. 3X

Fig. 8. An oblique row of ellipsoidal lesions. 5X

Figs.9 and 10. Irregularly scattered ellipsoidal and circular lesions. 5X.

Fig.11. Irregularly scattered longitudinal rows of lesions. 6X.

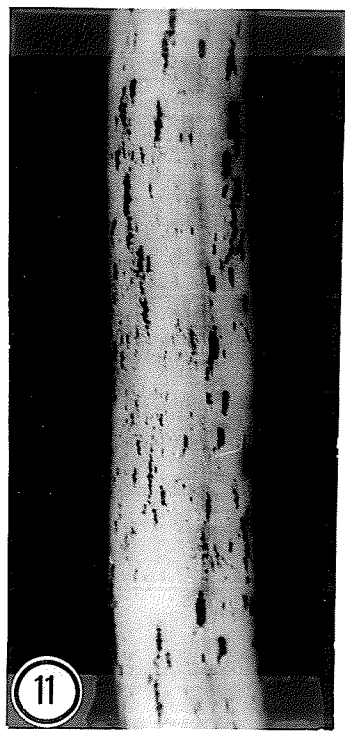
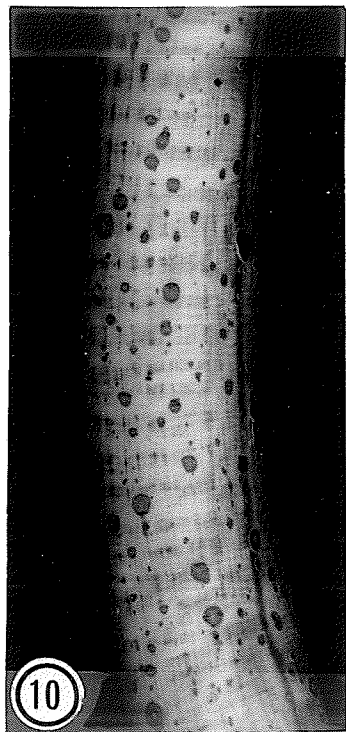
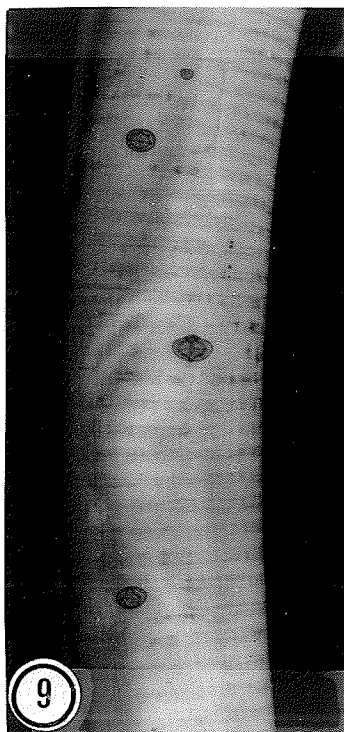
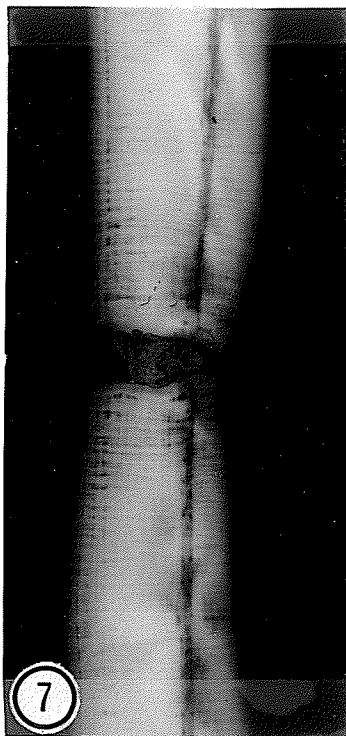
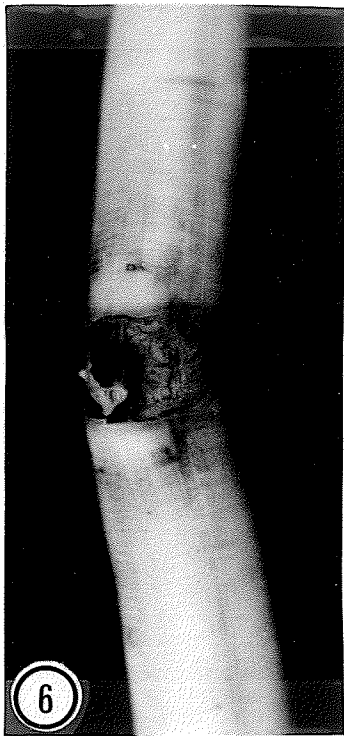


PLATE II

PLATE III.

Fig. 12. Large lesion, showing permanent bending
of the body. 15X.

Fig. 13. Circular lesions. 15X.

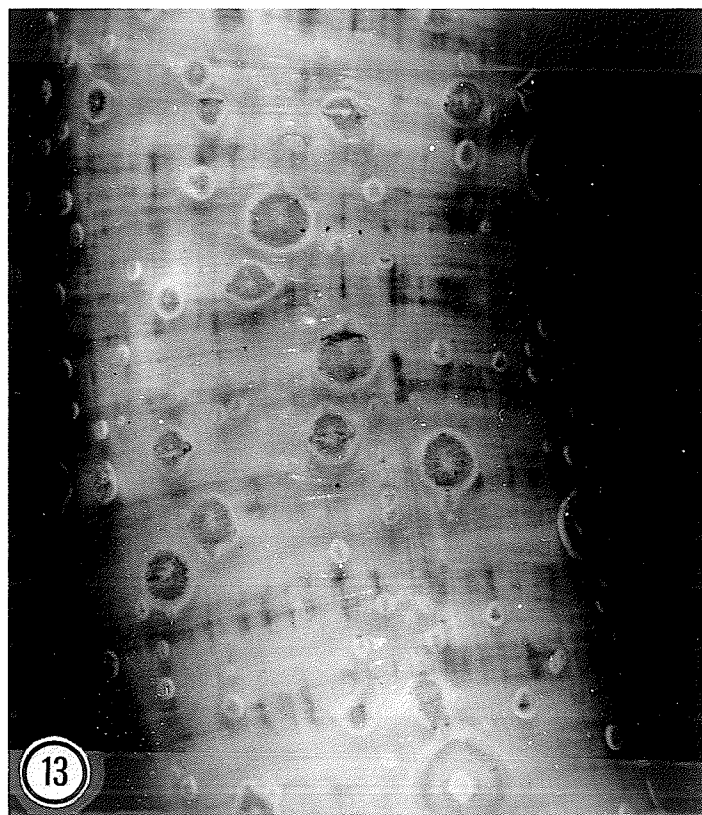


PLATE III

4. NORMAL STRUCTURE OF THE BODY WALL.

A study of the histopathology of dermomyositis in *Ascaris* necessitates a brief discussion of the normal structure of its body wall. As in other nematodes this wall consists of a cuticle, of an epidermis (hypodermis), and a single layer of muscle cells (Fig.15).

The cuticle covers the whole of the external surface and also lines the buccal cavity, oesophagus, rectum, cloaca, vagina, and excretory pore. The external cuticle, composed of proteins, with traces of fat and carbohydrates (14) is basically a three-layered structure (7,8,22,23), consisting of an outer cortex, a middle matrix layer and an inner basal layer. These three layers are subdivided into nine layers (2,3,4,7,8,38) (Figs.14-15). The cortex, consisting of an external vertically striated layer (4), and an internal one, is made of a loose network of fibers (38). Recent investigations with the electron microscope show that the cortex is covered by a thin osmiophilic membrane, probably lipoid, which is the main barrier to penetration of some anthelmintics (4,36). Beneath the cortex is the fibrillar layer, made up of tubular structures extending from the matrix to the cortex (4,38). The matrix or homogeneous layer is striated, these striations being continuous with the tubular structures of the fibrillar layer and the vertical striations of the external cortex (4). Beneath the matrix lies the very thin boundary layer, not shown in Figs. 14-15.

The next three layers are the so called fiber layers; their fibers run in spirals at about 70° to 75° to the long axis of the worm (33). The fibers of the outer layer are parallel to those of the inner one, and at an angle of about 40° to 45° to those of the middle layer, thus forming an intricate system of minute parallelograms. This system of fibers in conjunction with the somatic muscles allows for an increase of 10 - 15% in body length of the worm (20). The fiber layers stain darker blue with Mallory's counterstain than the adjacent layers of the cuticle (Fig.15). The basal lamella, the innermost layer of the cuticle, is in contact with the hypodermis; it is fibrillar and contains spaces connecting with the hypodermis the inner layers of the cuticle (38).

In summary, the important structural features of the cuticle are: the thin lipoid membrane covering the cuticle and acting as a defensive barrier, the pore canals connecting the hypodermis with the cortex, and the intricate fiber layers which allow for movement and extensibility of the body.

The hypodermis (stained red in Fig.15) is a syncytium containing fine fibers linking the muscle cells to the cuticle (21,38). The hypodermis is extended into the pseudocoelom forming four longitudinal chords running the length of the body. The dorsal and ventral chords contain nerves, the lateral chords, both nerves and excretory canals. Numerous nuclei are scattered throughout the hypodermis, the majority being found in the chordal areas. The

Key to Fig.14 EC, external cortex; IC, internal
cortex; F, fibrillar layer; M, matrix
(homogeneous layer); FL, fiber layers;
BL, basal lamella.

(Redrawn after Lee, 1966)

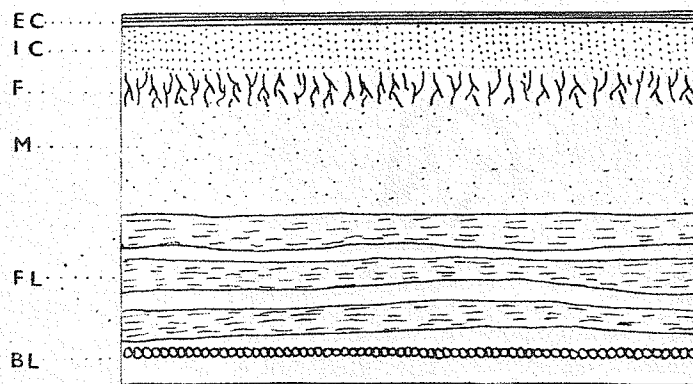
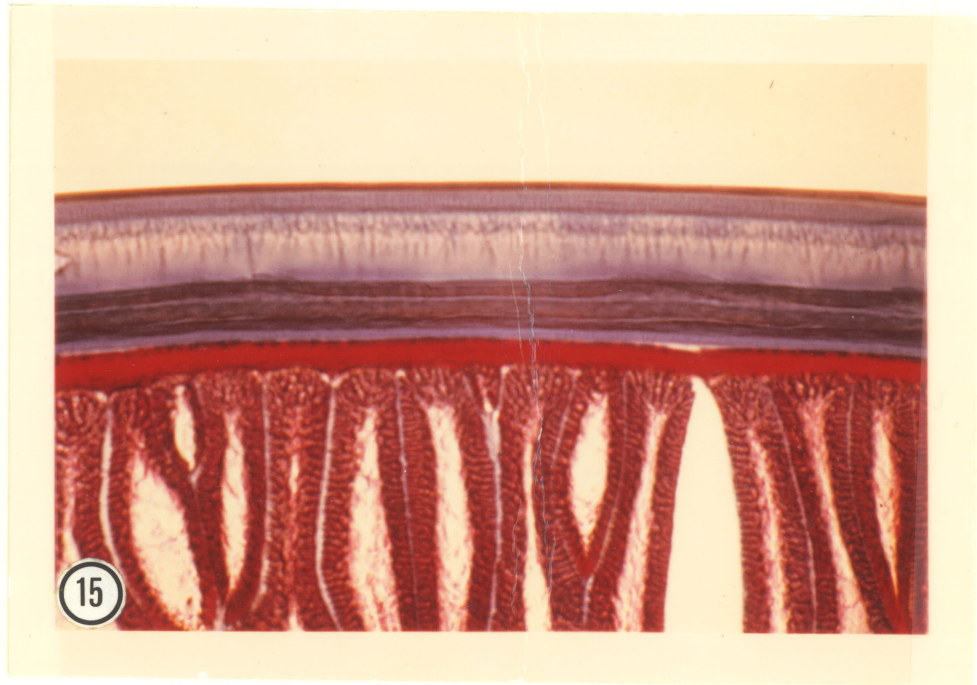


Fig. 14. Diagram showing a transverse section of the normal cuticle of Ascaris lumbricoides.

Fig. 15. Transverse section of a normal cuticle
of Ascaris lumbricoides. (Mallory). 250X



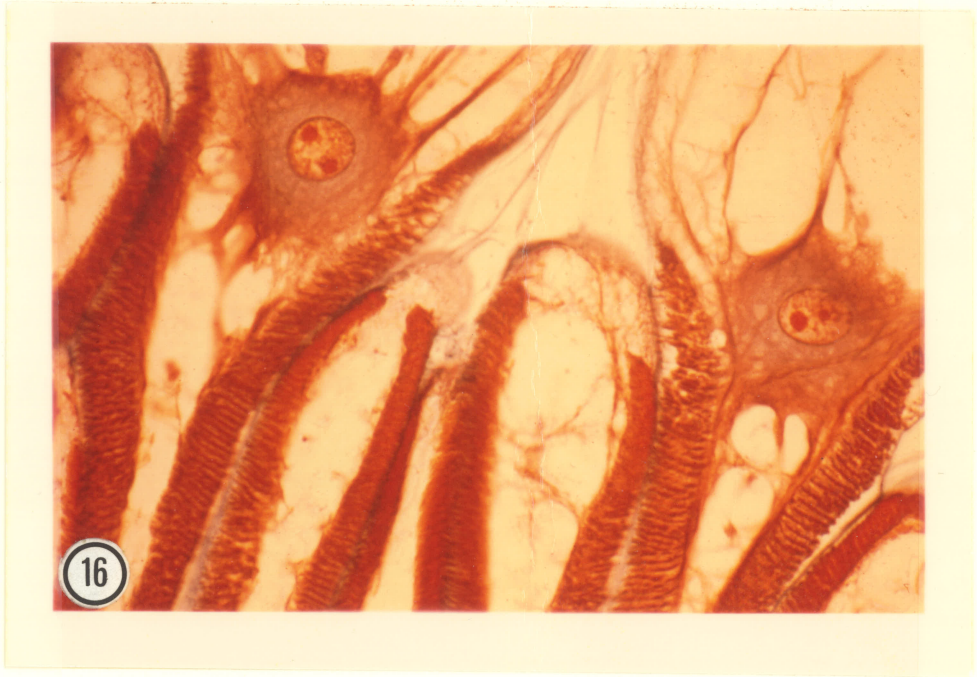
hypodermis is a region of high metabolic activity and a storage place for fat and glycogen (4).

The muscles of the body wall are coelomyarian and divided into four quadrants by the hypodermal chords. All are longitudinal and spindle shaped, there being no circular muscles in the somatic musculature of A. lumbricoides (7,8,21). Each muscle is composed of two parts, the sarcoplasmic bulb which extends into the pseudocoelom, containing the nucleus and giving rise to the innervation processes (Fig.16), and a contractile, fibrillar portion attached to the hypodermis (38). These longitudinal muscles act not against other antagonistic muscles but against the internal turgor pressure, (70 mm. Hg.), the so-called "hydrostatic skeleton" (20).

Contrary to popular belief the nematode cuticle is "alive" (10,23). There are two centres of protein synthesis in the body wall of Ascaris, the inner cortex of the cuticle and the hypodermis (1). Watson 1965, (38) suggested that precursors of cuticular material are transported from the hypodermis to all layers of the cuticle by a system of pore canals previously mentioned.

There is considerable growth of the cuticle in the adult Ascaris, the matrix layer increasing in thickness more rapidly than the fiber layers and both growing faster than the cortex (38). The material for this growth according to Bird 1957, (3) is secreted by the hypodermis, "Evidence obtained from this and other investigations (Bird and

Fig. 16. Transverse section through the muscle cells of A. lumbricoides showing muscle nuclei and innervation processes.
(Mallory) 250X



Deutsch 1958) points towards the cuticle being secreted by the lateral lines and subcuticle, rather than being a transformation or "condensation" as suggested by Chitwood and Chitwood (1937) of living material into dead material. The hypothesis is put forward without any direct evidence, that the cuticle of a zooparasitic nematode, such as A. lumbricoides is in a state of constant metabolic activity. At times, such as during moulting, there is increased activity and complex structural changes occur". Anya (1) in 1966 suggested that in some nematodes small molecules (amino acids and sugars) pass between the fibrils of the various layers of the cuticle and are synthesized into collagen fibrils in the outer layers, while large molecules and fibrils of collagen formed in the hypodermis pass into the basal layers to become incorporated in the fiber layers. These statements agree well with the observations of Metchnikoff (1891) (31) on the intensive growth of cuticle in Rhabditis encapsulated in the body of earthworms, and with the considerable thickening of the cuticle in dermomyositis of the pig ascarid (24,25,29,30). The cuticle of A. lumbricoides has two important functions, to protect the body against possible injury and to isolate the tissues of the body from the external environment.

5. HISTOPATHOLOGY OF THE LESIONS.

The smallest lesions studied are not accompanied by any appreciable changes in the hypodermis. In such lesions the basal lamella of the cuticle becomes three to five times thicker than normally, and has a tendency to compress the hypodermis and push it towards the pseudocoelom. Fig. 17 represents a transverse section through a small dermo-myositic punctata lesion. With Mallory's stain the pathological cuticle stains orange-red, the surrounding normal layers, blue. The damaged area of the cuticle is about 85μ wide and 20μ deep and extends into the fibrillar layer. The fiber layers (the dark band running through the middle of the cuticle) are unchanged. In the immediate vicinity of the lesion, the basal lamella is thickened from a normal 25μ to 75μ . This thickening extends almost a quarter of a millimetre on either side of the wound. Red stained fibers of the hypodermis penetrate into the thickened, scalloped basal lamella. The hypodermis, though slightly compressed, appears to be unchanged. At this state the muscle cells are not affected and the lesion seems to be superficial.

Fig. 18 shows a lineata type lesion almost one millimetre wide which has developed in the region of a lateral line. The cuticle near the middle of the lesion is deeply infolded, probably as a result of a longitudinal scratch by a sharp object in the intestinal contents. The fiber layers, stained orange in this section, end at the periphery

Fig. 17. A transverse section through a small,
superficial dermomyositic lesion.
(Mallory). 95 X

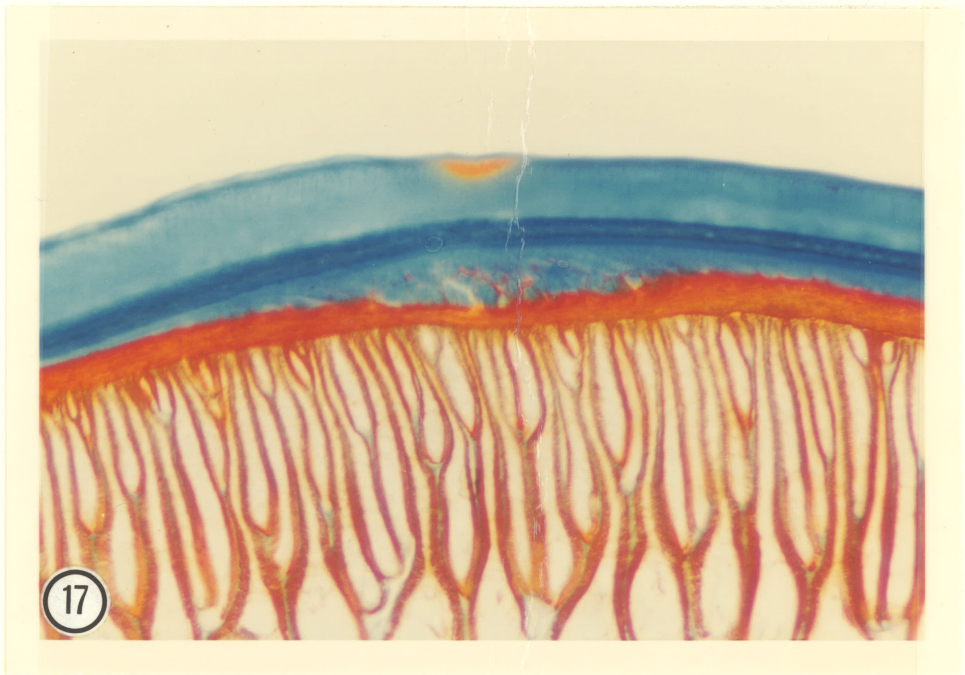


PLATE IV.

Fig. 18. A transverse section through a lesion about 1 mm. in diameter, showing the pathological cuticle, hypertrophied hypodermis and vacuolation of the muscle cells. (Mallory). 75X

Fig. 19. Photograph of the muscle cells in Fig. 18 taken at a higher magnification. 200 X

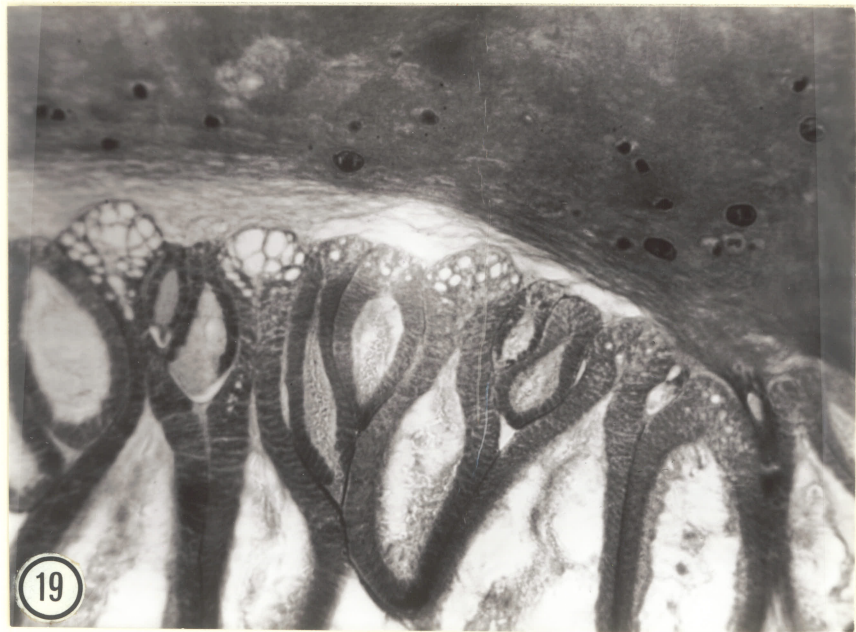
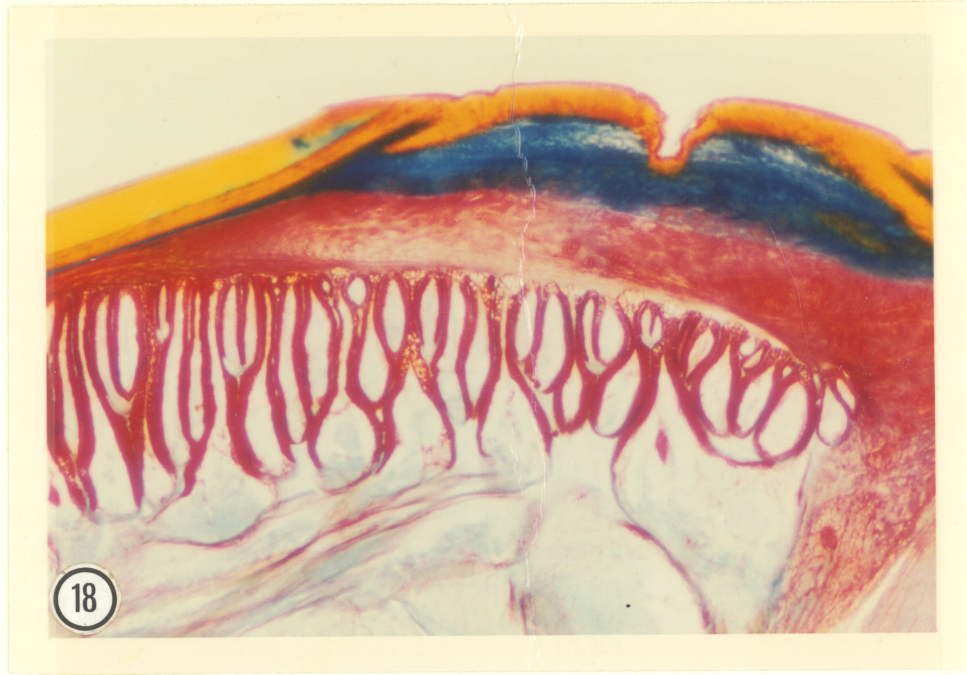
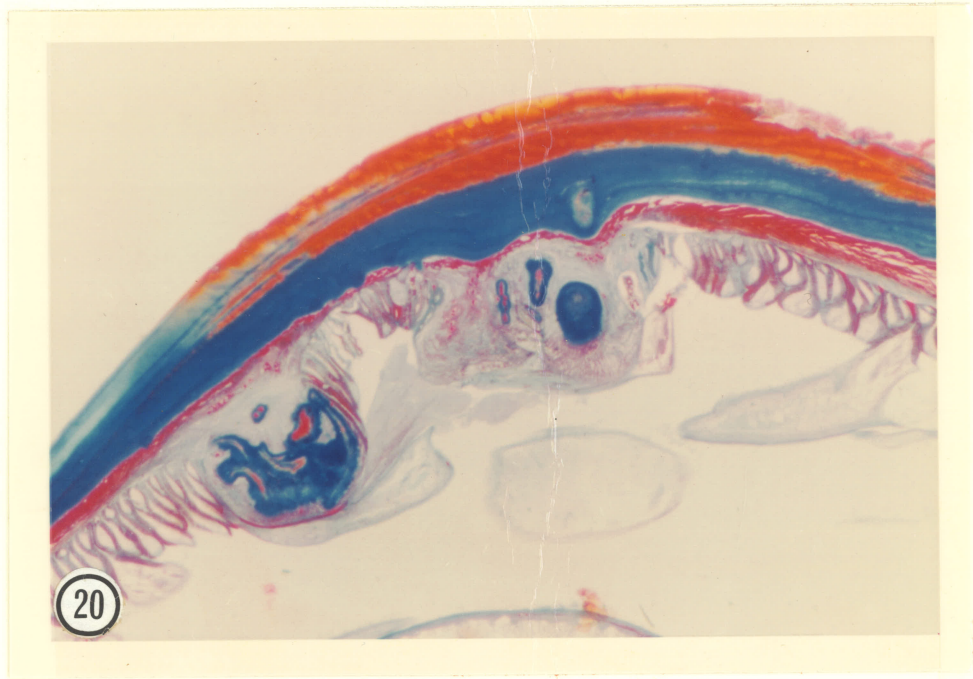


PLATE IV

of the lesion and are pushed slightly up and outwards by the new cuticle stained dark blue, which extends over the hypertrophied hypodermis and underneath the damaged cuticle. The new cuticle is about $115\ \mu$ thick, but becomes progressively thinner at the periphery of the lesion, where it is continuous with the basal lamella of the normal cuticle. The hypodermis and the proximal half of the lateral line are hypertrophied, the hypodermis being $85\ \mu$ thick, instead of the normal $15-20\ \mu$. The red stained fibrils of the hypodermis extend for a considerable distance into the pathological cuticle. Numerous nuclei are present in the hypodermis, the great majority being arranged in long rows parallel to its surface. At variance with the small lesion previously described, the muscle cells here show a pronounced vacuolation of their basal portions. In a few muscle cells the vacuoles spread a considerable distance towards the sarcoplasmic bulbs with no apparent change in the latter. Fig. 19 is a photograph of the muscle cells in Fig. 18 taken at a higher magnification.

A large lesion of the "rupia" type, extending a quarter of the circumference of the worm's body is shown in Fig. 20. The pathological cuticle, as in the previously described lesion, extends between the damaged normal cuticle and the hypodermis to the periphery of the lesion. The hypodermis under the central thickest portion of the new cuticle is very thin and clearly shows its fibrillar structure. Beneath the hypodermis and penetrating deeply into the musculature are two large structures $350\ \mu$ by $250\ \mu$ and

Fig. 20. A transverse section through a large lesion showing two peculiar structures containing accumulations of cuticular material. (Mallory). 75X



250 μ by 250 μ , which contains large accumulations of material whose structural and tinctorial properties are identical to those of the cuticle. Such structures were found only in one out of fifty five lesions examined histologically. The structure measuring 250 μ by 250 μ is a hypertrophied median line. It can be easily recognized by the innervation processes of the muscle cells attached to it. The normal median line on the opposite side of the body is only 70 μ wide near its internal end and is connected to the body wall by a long but narrow (10 μ) ridge of tissue. The affected median field is enormously enlarged and circular rather than long and narrow. At its base, where it is continuous with the hypodermis, there is a considerable multiplication of the nuclei, but some of them can also be found at the top of the median field. The centre of the median line is occupied by large concentric accumulations of cuticle-like material, whose periphery stains blue, and the center bright red. Some degenerate muscles adhere to the sides of the median field and are permeated by the fibrils of the hypodermis.

In the larger polyp-like structure measuring 350 μ by 250 μ , numerous nuclei, cuticular concretions and remnants of degenerate muscles can be clearly seen. The muscle cells between the two structures are highly degenerate.

A transverse section through the largest lesion (rupia) observed is represented in Fig. 21. The lesion is peculiar in several respects: first, it extends over half the

circumference of the body from one median line to the other and second, it shows an extensive vacuolation and partial liquefaction of the hypodermis. The entire lesion is up to 0.6 millimetres thick, thus about half the radius of the body. This extreme thickening and degeneration of the hypodermis extends over one quarter of the body circumference between a lateral line and one of the median lines. In the remaining portion (Fig.22), extending from the same lateral line to the other median line, the hypodermis is but slightly hypertrophied and is not vacuolated, the entire thickness of the lesion being only about 100 μ .

The highly hypertrophied and degenerate portion of the hypodermis is represented in Fig.21. The normal cuticle is present here only near the right margin of the lesion, where a wedge shaped, blue stained, pathological cuticle extends between the hypertrophied hypodermis and the remaining portion of the normal cuticle. Near the centre of the lesion, the pathological cuticle is from 100 to 150 μ deep. Its deeper portions are permeated by red stained fibers of the hypodermis. The superficial portion of the hypodermis is vacuolated and contains degenerate nuclei. Although the majority of the vacuoles are relatively small, some of them are 300 to 400 μ in diameter. Only the deep layer of the hypodermis adjacent to the muscle layer contains numerous nuclei (Fig. 23). Most of them are irregularly scattered in the hypodermis, some are arranged

in rows between the fibrils of the hypodermis. The underlying muscles are pushed deep into the pseudocoelom, but at variance with the conditions in lesions previously described, do not show any vacuolation. The lateral line is markedly hypertrophied and is one millimetre wide and one millimetre deep. It contains numerous nuclei mostly arranged in longitudinal rows.

The remaining portion of the lesion (Fig.22) between the hypertrophied lateral line and the opposite median line is covered with normal cuticle under which a deposition of pathological cuticle up to 150 μ thick is present. The inner surface of the pathological cuticle is scalloped and the fibrils of the hypodermis penetrate deeply into it.

PLATE V.

Fig. 21. A transverse section through a large dermomyositic lesion showing extensive hypertrophy and vacuolation of the hypodermis. (Mallory). 75X

Fig. 22. Left portion of the lesion shown in Fig. 21. (Mallory). 95X

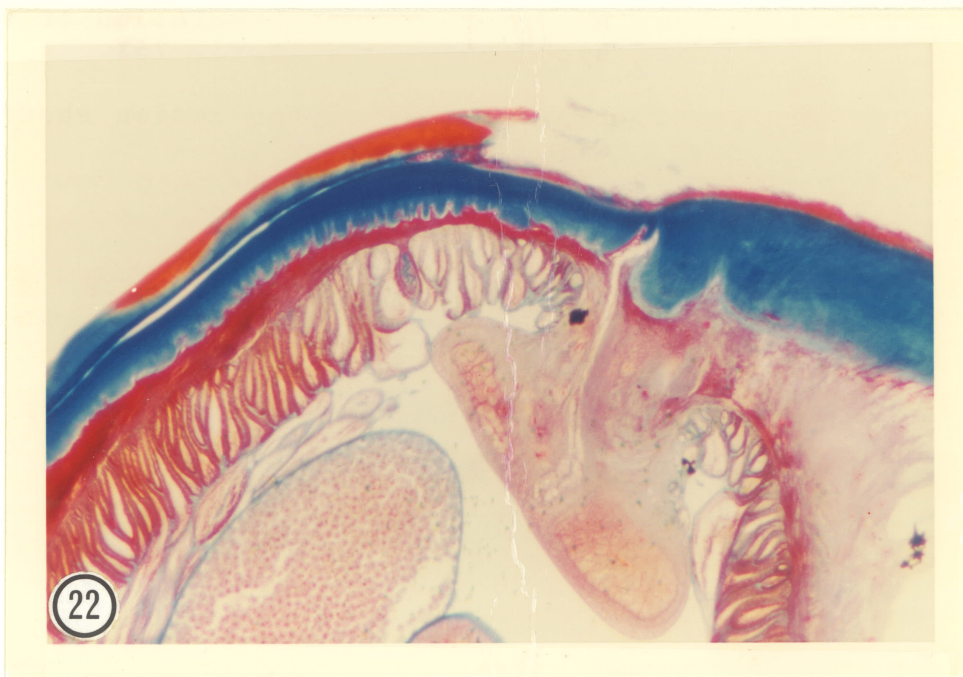
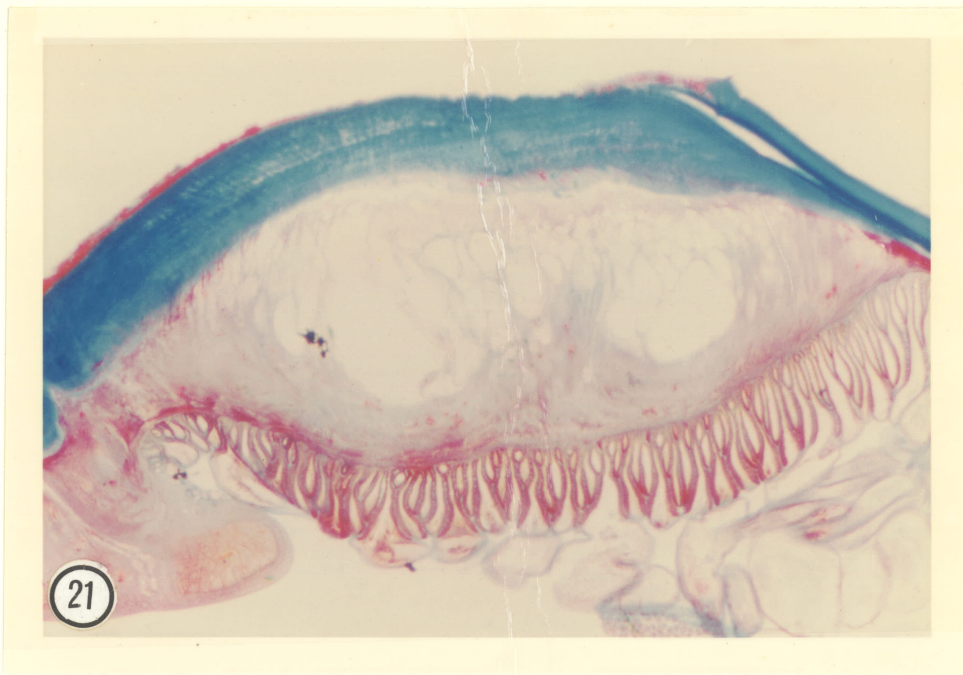
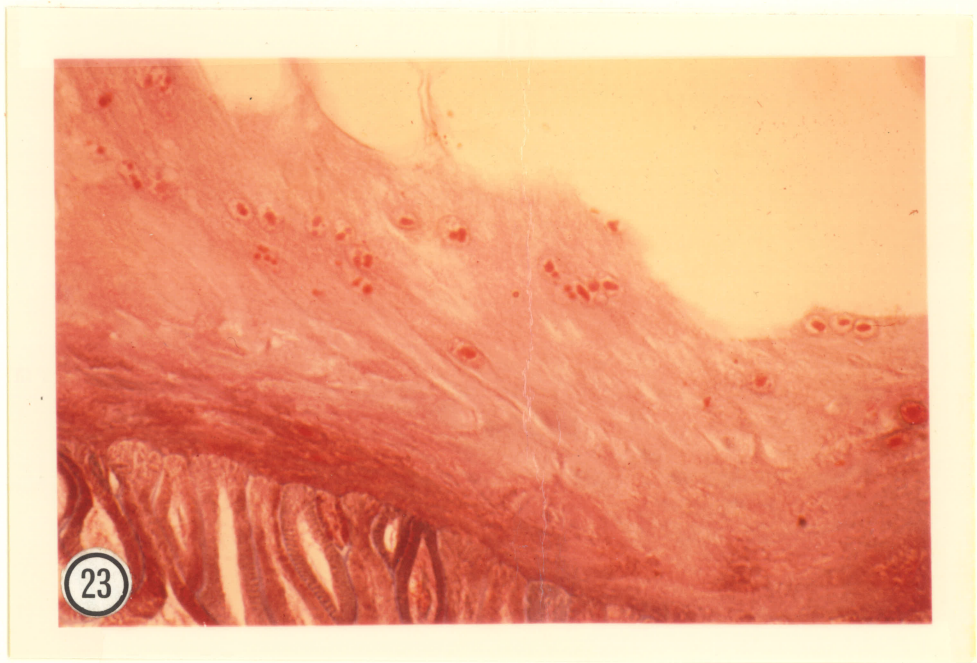


PLATE V

Fig. 23. Numerous nuclei in the hypodermis
of the lesion shown in Fig. 21.
(Mallory). 150X



6. MICROORGANISMS FOUND IN THE LESIONS.

Three different forms of microorganisms were found on the surface of the lesions or embedded in the pathological cuticle. Two of them were fungi, the third was a species of bacteria. In almost all cases colonies of bacteria were found in the lesion especially in the crevices of the affected area, whereas fungi were found in only three sections examined. The normal cuticle not affected by dermatomyositis did not contain any microorganisms, except for a few incidental bacteria adhering to its surface.

The fungi of the first type were ovoidal and subcylindrical cells, 6 to 10 μ long and 2 to 4 μ thick, arranged in short longitudinal rows. Almost the entire surface of the lesion shown in Fig. 21 was covered by them. They were especially numerous in the angles between the pathological cuticle and the margins of the normal cuticle (Fig.24), where they penetrated about 50 μ deep into the pathological cuticle. At least one long hypha growing from the surface of the cuticle was observed, (Fig.25). Some of the cells were in the process of budding.

The second type of fungus was found in the lesion characterized by cuticular concretions (Fig.20). These microorganisms were subspherical and measured from 2 to 6 μ in diameter. They were embedded up to 50 μ deep in the cuticle forming colonies from 20 to 40 μ in diameter (Fig.26).

PLATE VI.

Fig. 24. A transverse section of a dermomyositic lesion showing penetration of fungi into the pathological cuticle. (Mallory). 1200X

Fig. 25. Fungi with at least one long hypha growing from the surface of the pathological cuticle of a dermomyositic lesion. (Mallory). 1200X

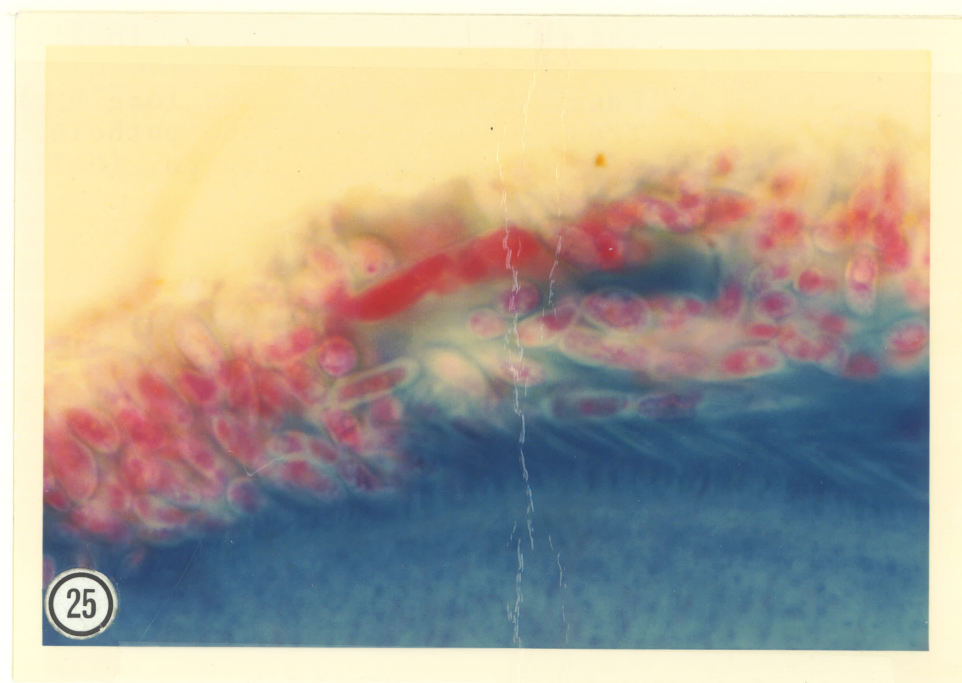
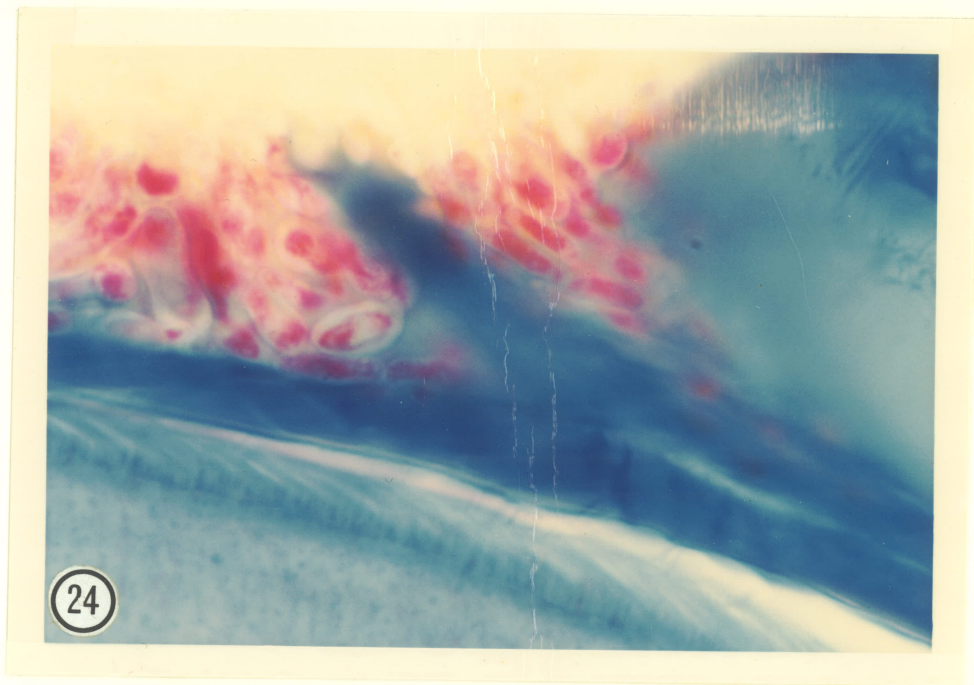


PLATE VI

PLATE VII.

Fig. 26. Another species of fungi embedded in the pathological cuticle of a dermomyositic lesion. (Mallory). 1200X

Fig. 27. A large colony of bacteria on the surface of a lesion. (Mallory). 1200X

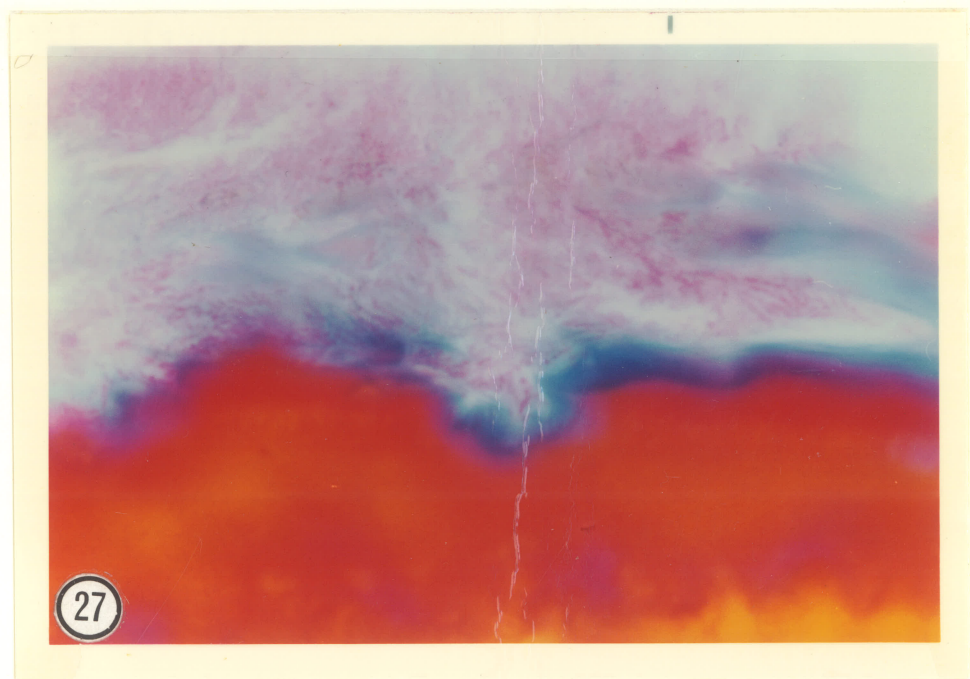
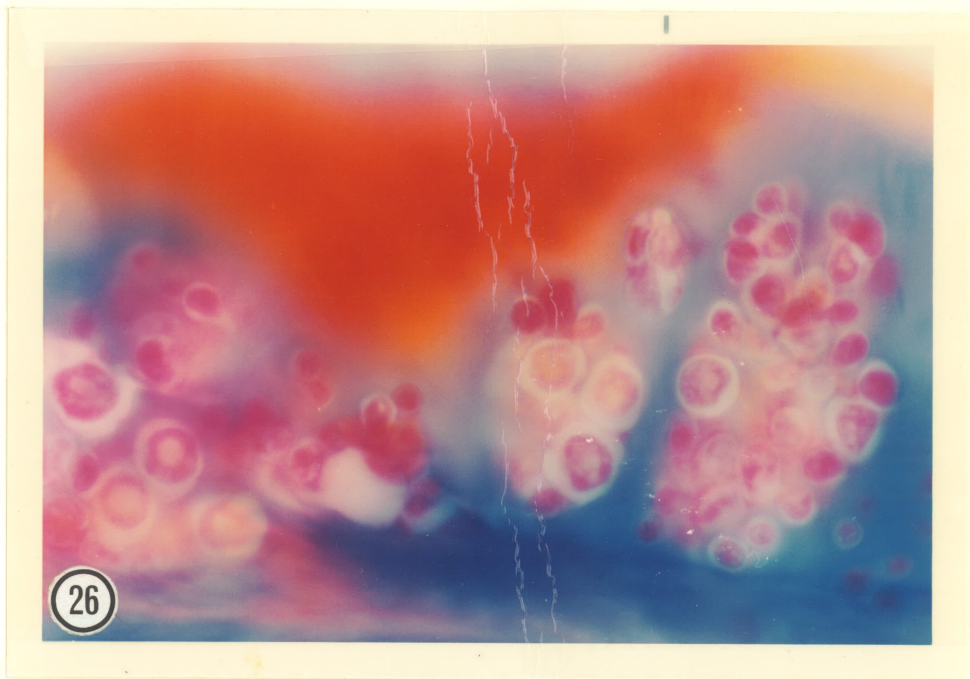


PLATE VII

The third kind of microorganism, bacteria, were found on the surface of the same lesion (Fig.20) where they formed a continuous layer 10 to 60 μ thick. These bacteria were rods about 0.5 μ in diameter and 2 to 4 μ long (Fig.27).

The identification of all three microorganisms necessitates their culturing which could not be attempted because the microorganisms were found in stained sections only. Such a study would be an interesting research topic.

IV. DISCUSSION

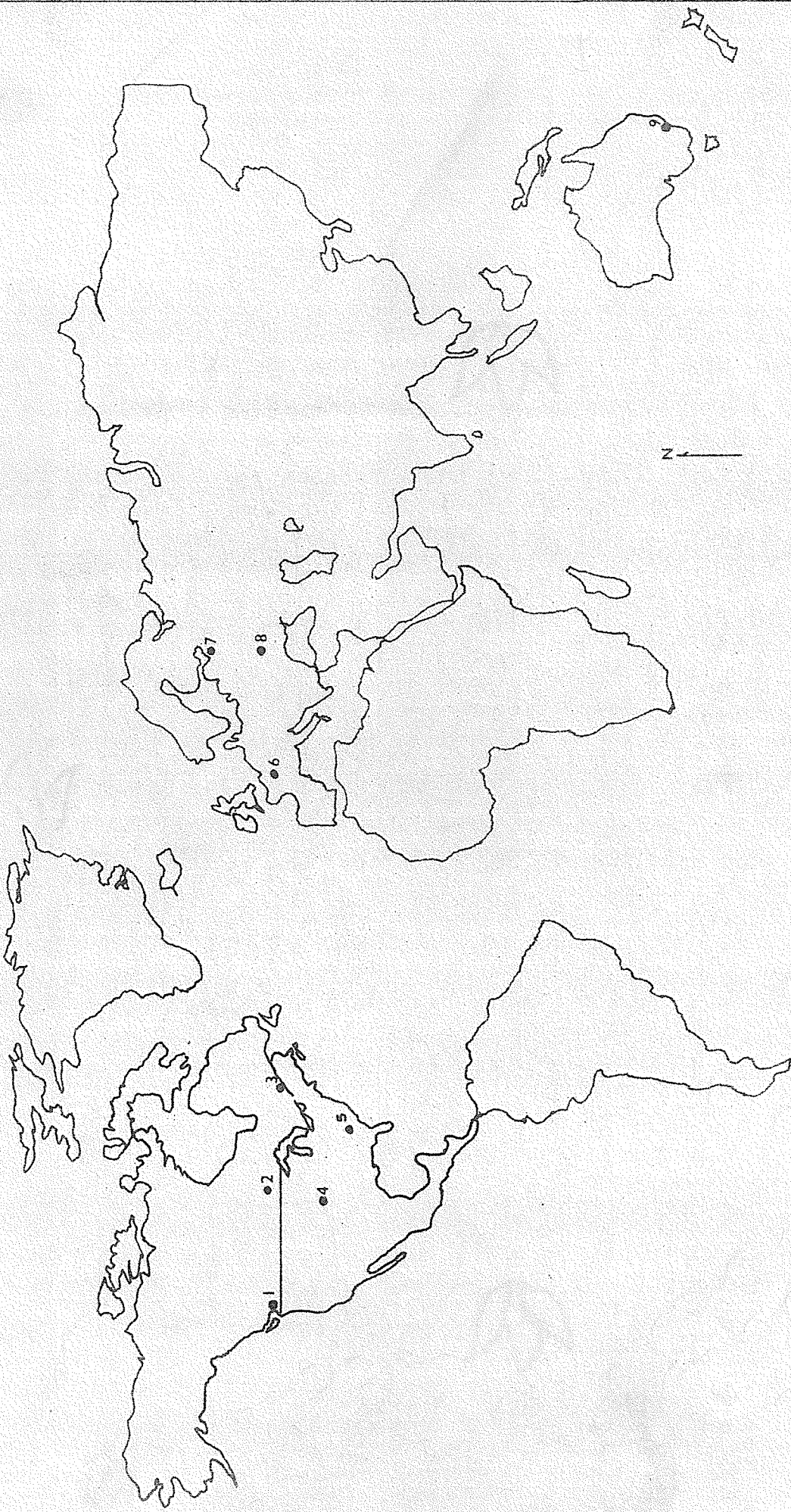
Dermomyositis of ascarids had not been previously recorded in Canada. I found this disease in approximately 2 per cent of the pig ascarids collected from Vancouver, B.C., Winnipeg, Man., and Montreal, Que.. The geographic distribution of recorded cases of dermomyositis of ascarids is represented in Fig. 28. This wide geographic scatter of the recorded incidence of dermomyositis suggests that this disease of ascarids is world wide in its distribution. Although no diseases of nematodes were found in Manitoba prior to the present work, a fungus predaceous on nematodes, Arthrobotrys superba Corda, was cultured from horse feces from Victoria Beach on Lake Winnipeg, Manitoba, by Bisby, Buller et al (5), as early as 1938. These authors also mention a subspecies of this fungus, Arthrobotrys superba oligospora.

The incidence of dermomyositis in ascarids from Manitoba, 1.9 ± 0.1 per cent, is considerably lower than any previously recorded. Weinberg and Keilin in 1912 working in Paris, found that of 196 horse ascarids examined 130 had lesions (66.3%). Lubinsky in 1931 (24) in Kiev, Ukraine observed that 94.3 per cent of the ascarids from pigs, but only 2 per cent of those from man, had the disease. The incidence of dermomyositis in nematodes from horses was: in Ascaris equorum 89 per cent, Oxyuris curvula 1.94 per cent. In Toxascaris limbata, the dog ascaris

Fig. 28. The geographical distribution of
dermomyositis of ascarids.

Key to map.

1. Vancouver, B.C.
2. Winnipeg, Man.
3. Montreal, Que.
4. Nebraska, U.S.A.
5. Georgia, U.S.A.
6. Paris, France.
7. Leningrad, Russia.
8. Kiev, Ukraine.
9. Sydney, Australia.



THE WORLD

the incidence was 1.91 per cent and in Heterakis perspicillum, the chick ascarid, 10 per cent. Dermomyositis was also recorded by Lubinsky in Strongylus equinus and S. vulgaris from the large intestine of horses. The percentage of occurrence was not recorded because of the difficulty of distinguishing, without histological examination, between small dermomyositic lesions and solidified secretions of the bursal glands of males. Cleland and Johnston (9), Manter (28), Pavlovskii (32) and Stewart and Godwin (34,35) provided no data on the incidence of dermomyositis.

The present research revealed some seasonal variation in the incidence of dermomyositis in Manitoba. It is lowest, 1.5%, in the spring (March, April, May), increases in the summer (June, July, August) to 1.9%, and reaches its peak, 2.2% in the autumn (September, October, November) to decrease again in the winter (December, January, February) to 2.1%. Due to production line methods of processing pigs in the packing houses, it was impossible to determine if there were any seasonal fluctuations in the numbers of worms in Manitoba.

In my material female worms outnumbered the males more than 2 to 1 (9400 to 4276). The incidence of dermomyositis in females was almost twice that in males, 2.1 ± 0.2 and 1.3 ± 0.2 per cent respectively. This may depend on the larger size and thus greater surface area of the females. From the sample of 100 worms the average surface area of

the females was 3470 square millimetres, compared with 1536 square millimetres for males, thus almost 2.3 times greater.

Many kinds of microorganisms were recorded from ascarids with dermomyositis. Weinberg and Keilin in 1912 (39) mistakenly identified large hypodermal nuclei of horse ascarids as protozoa. They also observed large cocci which they thought played a secondary role as the cause of the disease. Manter in 1929 (28), described masses of bacteria growing from the thickened base of the lesions as resembling Clostridium welchii. Pavlovskii in 1934 in Leningrad (32), believed that the lesions were caused by accumulations of bacteria (Bacterium ascaridianum) which eroded the cuticle. Stewart and Godwin in 1959 (34,35), identified three microorganisms from dermomyositic lesions: Escherichia coli, Candida sp. and Pseudomonas sp.. They were able to produce lesions of the cuticle experimentally with cultures of Pseudomonas sp., but were unsuccessful with E. coli, Candida sp., or C. welchii. In my material three kinds of microorganisms were present on the pathological cuticle. These microorganisms were either on the surface of the cuticle or embedded in it. Two of them were fungi and one a bacterium. In one lesion both a fungus and a large colony of bacteria were found.

Epps et al in 1950 (13), reported finding three types of bacteria in ground-up normal ascarids: gram-positive cocci, gram-negative rods producing small, elevated colonies, and

gram-negative coliform bacilli producing large colonies. Bird and Deutsch in 1958 (4), isolated gram-negative motile rods, probably Pseudomonas aeruginosa, from the surface of the normal cuticle of A. lumbricoides.

A thorough examination of cutaneous lesions is extremely important. Heinze in 1937 pointed out that bacterial accumulations on the skin of nematodes may simulate cutaneous lesions, on the other hand they may be associated with such lesions. Perforations of the skin by fungal hyphae may also sometimes be mistaken for normal structures, eg. the pores of the areolar canals. Heinze described stellar structures on the skin of "Gordius stellatus" which he regarded as lesions of parasitic origin. See Dollfus, Fig. 126, page 148 (11).

Several views as to the origin of dermomyositic lesions have been expressed. Cleland and Johnston (9), believed that injuries to the cuticle of A. suilla might have been inflicted by "Gigantorhynchus gigas" = Macracanthorhynchus hirudinaceus (Pallas, 1781) which were found in considerable numbers together with the ascarids in the pig's intestine. Lubinsky (24), suggested that rough or sharp objects in the host's intestine scratch the cuticle. I believe that another source of injury may be the spicules of the males scratching the cuticle especially when the worms are numerous. I did not find any other parasites while collecting ascarids for this project. Lubinsky (26) informs me that M. hirudinaceus was found but seldom in the Ukraine and

that ascarids with numerous dermomyositic lesions were common in pigs free of this parasite. The presence of various microorganisms in the lesion does not contradict Lubinsky's contention that dermomyositis arises as a result of scratches. It is obvious that the wounds of ascarids may be infected by various microorganisms. Vago in 1963 (37), stated that, in insects, the hemolymph coagulum covering the wound favours the growth of certain fungi, whereas open wounds are more often infected by bacteria. My material falls naturally into two groups, one with small numbers and one with large numbers of lesions. In both of these groups the lesions are probably caused by a combination of injury to the cuticle and subsequent infection by bacteria or fungi or both. The absence of worms with 20 to 165 lesions is remarkable but not easily explained. It may be conjectured, that in the first group, few lesions may be caused by the swallowing by the host of a few sharp objects, for example gravel, or small amounts of sand, whereas multiple lesions may depend on the ingestion of large amounts of sand.

All animals possess defense mechanisms against injury such as the penetration of bacteria, parasites or non living foreign bodies into their tissues. Metchnikoff in 1893 (31) observed that, when a foreign body penetrates into the body cavity of the larva of Astropecten, a sea star, the mesodermal cells move toward it, and surround it, forming a plasmodium. Thus the first reaction to injury in

this animal, which has no vascular system, is the migration of mesenchymal cells towards the foreign body with subsequent phagocytosis or encapsulation.

In the vertebrates and other higher animals the process of inflammation is more complex. When tissue is injured leucocytes, mainly neutrophils and monocytes, migrate from the blood into the tissue. These wandering phagocytes are the organisms first line of defense. Their migration is facilitated by the dilation of the capillaries around the injured area. Numerous fixed phagocytes found in the liver, spleen, lymph nodes and bone marrow, and in other organs, are collectively known as the reticulo-endothelial system, RES (16). However, in the local inflammatory reaction the wandering leucocytes are more important than the reticulo-endothelial cells.

The nematodes lack a vascular system and have a few cells which are probably phagocytic. In Ascaris lumbricoides, for example, the pseudocoelom is partially lined by Goldschmidt's "isolation tissue" which is of mesodermal origin. A single large nucleus is dorsal to the oesophagus between the nerve ring and excretory pore. From the cytoplasm surrounding this nucleus a delicate spongy material extends to fill the pseudocoelom. It also covers the muscle cells and penetrates between them to the hypodermis. It also produces the pseudocoelomic membrane which covers the lateral and median chords, the intestine, and the gonads (7,8).

A. lumbricoides has only four fixed stellate cells, coelomocytes, situated near the lateral lines in the anterior third of the body. Though of mesodermal origin, they are not part of the isolation tissue. Each cell has a large nucleus and a protoplasm produced into numerous branching processes with spherical bodies at their ends. Chitwood and Chitwood (7,8) injected India ink and Escherichia coli into A. lumbricoides and found these cytoplasmic extensions covered with India ink particles or with bacteria. According to these authors the coelomocytes are "absorptive or phagocytic cells, comparable to the fixed histiocytes of vertebrates. As the body fluid flows by them it is assumed that they purify it in some manner." The pseudocoelomic fluid of nematodes is cell free (15). Coelomocytes of ascarids can thus be compared to the reticulo-endothelial system of vertebrates.

In dermomyositis of ascarids the tissue reaction to the injury is expressed in the hypertrophy, and sometimes degeneration, of the hypodermis which may increase in thickness 7 or 8 times. This hypertrophied hypodermis produces layers of pathological cuticle up to 150 μ thick. The adjacent muscle cells may become vacuolated (Figs.18-19). It is obvious that in A. lumbricoides the main role in the defense against injury and infection is played not by mesodermal cells but by the hypodermis, which is of ectodermal origin. It may be of interest that, when Lumbricus is invaded by Rhabditis, this nematode is surrounded by phago-

cytes of the host and, according to Metchnikoff (31) "secretes layers which form, not a true cyst, but a supplementary cuticle which frequently becomes of extraordinary thickness." Metchnikoff also stated that nematodes "protect themselves by the secretion of tough membranous cuticles, resembling in this feature the plants, the cells of which are likewise protected by thick, resistant membranes."

In some higher animals, such as vertebrates and arthropods, the epithelium also participates in the inflammatory reaction of the skin, especially against small foreign bodies. Garshin (17,18) described the process of epithelization of foreign body granulomas of the subcutaneous tissue in rabbits. It is also known, that the larvae of the mosquito, Aedes aegypti (L.) (6,40), invaded by rhabditoid nematodes (DD136), encapsulate them as a result of the usual inflammatory reaction. Host epithelial cells may grow around the capsule isolating the foreign body which may be eliminated in the next moult. In the great majority of animals the epithelium plays only a secondary role in the process of inflammation, though the epidermal cells do become activated during wound healing (Wigglesworth 1937,1957) (41,42). In nematodes which have no wandering leucocytes, the reaction of the epithelium to injury is of primary importance. The enormous increase in the thickness of the hypodermis and the excessive production of the pathological cuticle may be regarded as an attempt to produce a more or less impenetrable wall between the living tissues of the

nematode and the invading microorganisms.

The study of dermomyositis and of similar pathological processes in nematodes is interesting from the standpoint of comparative pathology and could be a fruitful field of research.

V. CONCLUSIONS

The present research has resulted in the following findings:

1. Dermomyositis in A.lumbricoides from pigs occurs in Manitoba, British Columbia and Quebec. This is the first record of this disease of nematodes in Canada.
2. The average incidence of dermatomyositis, based on the examination of 14,441 ascarids, is 1.9 ± 0.1 per cent.
3. The incidence of dermatomyositis in Manitoba varies with the season, being lowest, 1.5 ± 0.2 per cent, in the spring (March, April, May) and highest, 2.2 ± 0.3 per cent, in the autumn (September, October, November).
4. Dermomyositis occurs more frequently in females, 2.1 ± 0.2 per cent, than in males, 1.3 ± 0.2 per cent, the difference being significant at the 0.01 level.
5. Lesions were scattered randomly over the body surface, no area being particularly affected.
6. Vacuolation of the hypodermis and the basal portions of the muscle cells found in lesions examined histologically were not recorded previously.
7. Three different kinds of microorganisms were found in the lesions. A bacterium produced large colonies on the surface of the lesion. The other two types of microorganisms were fungi, found either on the surface or embedded in the pathological cuticle of the lesion.

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APPENDIX A.

NUMBERS OF LESIONS PER QUARTER OF BODY SURFACE

Anterior quarter	Second quarter	Third quarter	Posterior quarter
48	53	37	20
56	123	73	28
69	126	100	53
108	149	126	96
130	182	175	108
167	182	234	196
172	186	273	212
216	278	280	233
224	344	286	240
226	353	323	260
250	378	356	268
258	441	358	270
260	448	431	315
288	455	463	320
290	463	483	342
295	475	509	347
310	502	511	359
372	528	512	370
409	551	525	412
410	560	533	418
451	572	600	425
462	576	638	448
531	592	647	460
581	601	663	540
585	648	684	560
648	658	686	581
658	741	784	582
668	743	787	585
700	781	819	592
745	865	864	630
812	900	906	644
826	931	1026	690
828	950	1050	702
920	1008	1127	735
936	1024	1183	918
954	1060	1197	923
1044	1122	1305	1005
1107	1155	1324	1125
1123	1274	1335	1151
1130	1290	1504	1170
1196	1350	1666	1228
1460	1422	1800	1263
1520	1482	1833	1351
1843	1729	1859	1581
1867	1872	1887	1842
2105	1977	2070	2010

$\bar{X} = 658$
N = 46

$\bar{X} = 741$

$\bar{X} = 800$

$\bar{X} = 622$

NUMBERS OF LESIONS PER SQUARE CENTIMETRES OF BODY SURFACE

Anterior quarter	Second quarter	Third quarter	Posterior quarter
81	64	76	78
161	121	176	168
108	52	94	120
78	116	125	157
36	70	65	68
108	123	139	98
9	7	5	3
141	241	256	350
36	56	35	34
146	130	150	162
53	71	77	22
71	74	86	115
258	178	158	113
38	24	13	5
57	55	57	41
165	153	160	97
67	28	38	47
183	151	125	154
160	122	134	106
124	112	91	79
38	53	56	71
71	70	71	64
70	76	58	67
40	45	33	48
82	56	78	44
21	12	35	45
8	11	7	6
143	88	97	89
25	36	29	34
120	84	75	65
43	46	44	43
93	74	95	90
47	46	56	30
10	23	19	21
192	165	156	206
95	134	109	149
85	88	110	130
181	118	120	132
51	89	69	77
94	107	149	118
155	115	158	119
72	38	48	67
110	80	69	69
131	102	121	135
32	46	42	60
114	89	95	148

$\bar{X} = 91$

$\bar{X} = 83$

$\bar{X} = 88$

$\bar{X} = 90$

N = 46

APPENDIX B.

CLASSIFICATION OF ASCARIS LUMBRICOIDES

The history of the systematics of "vermes" or "helminths" is extremely complicated. Indeed, animals designated as helminths are very diverse and do not form a natural group. This is why the name "vermes" is absent from recent literature.

Rudolphi in 1808 divided the "vermes" into several groups, Nematodea, Acanthocephala, Trematoda, and Cestodea. In 1851 Vogt placed the flatworms and nemertines into the group "Platyelmia" and the gregarines, acanthocephalans, nematodes and gordiaceans in the group "Nematelmia". Gegenbaur in 1859 changed "Nematelmia" to Nemathelminthes, a term still widely used today. Diesing in 1861 changed Rudolphi's term Nematodea to Nematoda.

The position of Ascaris lumbricoides in Chitwood's classification of the nematodes is as follows:

Phylum	Nematoda	(Rudolphi, 1808) Diesing, 1861
Class	Secernentea	(Von Linstow, 1905) Dougherty, 1958
Order	Ascaridida	Chitwood, 1959
Suborder	Ascaridina	Chitwood and Chitwood, 1937
Superfamily	Ascaridoidea	Railliet and Henry, 1915
Family	Ascarididae	Blanchard, 1896
Subfamily	Ascaridinae	Lane, 1923
Genus	<u>Ascaris</u>	Linnaeus, 1758
Species	<u>lumbricoides</u>	Linnaeus, 1758

Synonyms:-

Ascaris gigas GOEZE, 1782

Ascaris suum GOEZE, 1782

Ascaris suis GMELIN, 1790

Fusaria lumbricoides hominum ZEDER, 1800

Fusaria lumbricoides suis ZEDER, 1800

Fusaria lumbricoides suum RUDOLPHI, 1809

Lumbricus teres hominis RUDOLPHI, 1809

Ascaris gigas hominis RUDOLPHI, 1809

Ascaris gigas suis RUDOLPHI, 1809

Ascaris ovis RUDOLPHI, 1819

Ascaris suilla DUJARDIN, 1845

Ascaris bifaria BAIRD, 1853

Ascaris lumbricoides was also known by its pre-Linnean name of
Lumbricus teres TYSON, 1683

There are several views as to the position of the nematodes among the protostomians and these views vary greatly. Whereas some authors regard the nematodes as a phylum, for example Chitwood and Chitwood (7,8), others regard it as a class; Yorke and Maplestone in 1926 (43) place it as a class in the phylum Nemathelminthes, Hyman in 1951 (21) and Goodey in 1963 (19) as a class in the phylum Aschelminthes.

APPENDIX C.

CHRONOLOGY OF THE STUDY OF DERMOMYOSITIS

- 1912 Weinberg, M. and Keilin
(Paris, France) A disease of Ascaris megaloccephala manifests itself in the appearance of yellow brown plaques on the cuticle was described under the name of dermomyositis.
- 1912 Cleland, J.B. and T.H. Johnston
(Sydney, Australia) Small injuries on the cuticle of Ascaris suilla and the process of their healing was described.
- 1929 Manter, H.W.
(Nebraska, U.S.A.) A description of yellow "sores" from microscopic to 1.5 mm. in diameter in pig ascarids.
- 1931 Lubinsky, G.
(Kiev, Ukraine) Dermomyositis found in Ascaris lumbricoides from both man and pigs and in various nematodes of horses, chickens and dogs.
- 1932 Lubinsky, G.
(Kiev, Ukraine) A description of the histopathology of dermomyositis in pig ascarids.
- 1934 Pavlovskii, E.N.
(Leningrad, Russia) Pigmented spots on the cuticle of ascarids produced by the accumulation of bacteria (Bacterium ascaridianum).
- 1959 Stewart, T.B. and H.J. Godwin (Georgia, U.S.A) In vitro infection of ascarids with Pseudomonas sp. shown to cause lesions similar to dermomyositis.
- 1966 McKinnon, G.A. and G. Lubinsky
(Winnipeg, Canada) A report on the occurrence and geographical distribution of dermomyositis of pig ascarids in Canada.

THE OCCURRENCE OF DERMOMYOSITIS OF ASCARIDS IN CANADA

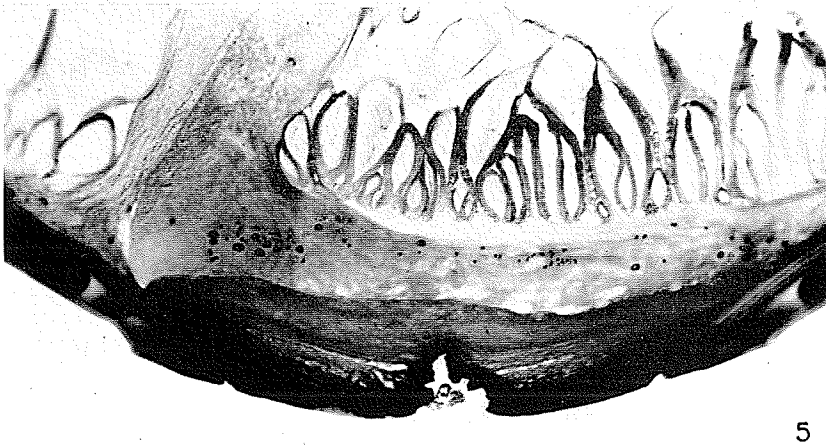
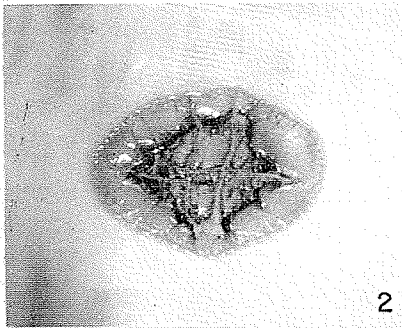
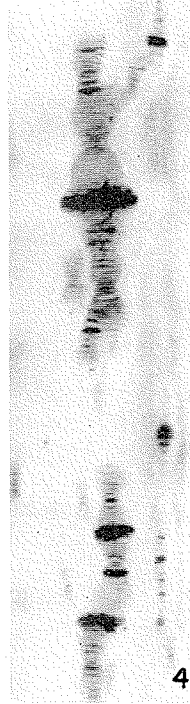
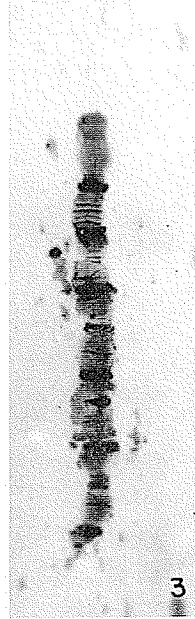
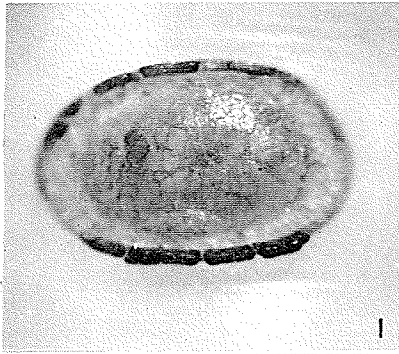
G. A. MCKINNON AND G. A. LUBINSKY

Weinberg and Keilin (6) described the dermomyositis of horse ascarids from Paris, France, in 1912. In 1929 Manter (3) noted dermal lesions in pig ascarids from Nebraska, U.S.A. In 1931 Lubinsky (1) in Kiev, Ukraine, found dermomyositis in ascarids from both pigs and man, in *Parascaris equorum*, *Oxyuris curvula*, *Strongylus equinus*, and *Strongylus vulgaris* from horses, *Toxascaris limbata* from dogs, and *Ascaridia perspicillum* from chickens. Later (2) he described the histopathology of the dermomyositis of pig ascarids. Recently Stewart and Godwin (4, 5) found this disease in pig ascarids from Tifton, Georgia, and produced the disease experimentally both in vivo and in vitro. Dermomyositis of nematodes was not previously recorded from Canada.

We examined 11,657 pig ascarids from Winnipeg, 765 from Vancouver, and 2019 from Montreal, and found dermomyosites in 1.9, 2.2, and 1.3% of the worms respectively, an average of 2%. Females were affected more frequently than males. The lesions occurred mainly on the anterior portion of the body, though about 40% of ascarids affected had multiple lesions scattered over the entire body surface.

Single lesions were mostly ellipsoidal, with their long diameters at right angles to the body axis (Figs. 1 and 2). Multiple lesions developed mostly

PLATE I



FIGS. 1-5. Lesions in *Ascaris lumbricoides*. Figs. 1 and 2, ellipsoidal, $\times 40$. Figs. 3 and 4, linear, $\times 30$. Fig. 5, section through a large dermomyositic lesion, $\times 70$.

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along longitudinal scratches and were transversally striated, probably as a result of transverse cracking of the cuticle (Figs. 3 and 4). Histologically all larger lesions show a pronounced hypertrophy of the syncytial hypodermis, with degeneration of the basal portions of muscle cells and deposition of thick layers of pathological cuticle (Fig. 5).

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6. WEINBERG, M. and KEILIN. 1912. Une maladie de l'*Ascaris megalcephala*. *Compt. Rend. Soc. Biol.* **73**, 260-262.

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