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AN INVESTIGATION OF THE PATHOLOGY OF "GROUSE DISEASE."

A REPORT OF THE INVESTIGATIONS UNDERTAKEN FOR THE GROUSE DISEASE INQUIRY.

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Introduction.

THE fact that the lower animals are subject, like man, to diseases does not obtrude itself upon our notice, probably because they often hide themselves when ill, and creep into some corner to die, and perhaps because they have less aptitude for expressing their sufferings. However this may be, they bear so generally the aspect of perfect health that when attacked by a serious epizootic disorder, the latter gets dubbed with the name of the species it attacks; and so one hears of silk-worm disease, horse sickness, or swine fever, as though they were the only diseases from which these species suffer.

The grouse, like other animals, suffers, doubtless, from a variety of diseases and disorders, but one of these, it is held, so far exceeds all the rest in importance that it has earned for itself the name of grouse disease. Sportsmen and gamekeepers bear undivided testimony to the existence of this epizootic, which appears with varying severity and regularity in Spring and Autumn; but it is not easy to be certain that in "grouse disease" we have to do with a specific infectious disease, as is generally assumed, or merely with a concatenation of disastrous consequences set in train by unusual privation, due perhaps to bad weather. Still less easy is it, in the case of any given bird, to tell whether it is suffering, or has suffered from, "grouse disease" or no, especially at a time when birds are dying in unusual numbers; for if we agree that there is a specific infection, we must admit also that privation claims its toll, and which of the two our bird is suffering from is not easy to decide, even after it is dead, for grouse disease has no characteristic symptoms, or very obvious macroscopic lesions.

Our work has been done both with diseased and healthy birds. The former were caught by the keepers in a feeble or dying condition, at times when dead birds were being picked up in considerable numbers on the moor, and it was consequently believed that "grouse disease" was prevalent. But the mortality then was, we believe, not very great, in fact insignificant when compared with the really bad years such, for example, as 1873, and it is, we suppose, just possible that we never came across the genuine epizootic grouse disease at all.

The diseased birds which were subjected to bacteriological examination were nearly all caught alive and brought or sent to the temporary laboratory. Some, of course, died on the journey, but in only a few preliminary instances were cultures made from the latter,

and then only when it was exactly known when the bird was last seen alive.

In addition to these birds from which cultures were made many others, which were picked up dead, were examined for lesions and gross parasites. As will be easily understood the difficulty of obtaining diseased birds alive was very great, and the number investigated therefore very small.

The control observations on normal birds were more numerous; for, through the kindness of Sir Richard Graham, Lord Lovat and others, we had no difficulty in obtaining as many as we wanted. Some few of these were examined in Scotland, but the great majority were received alive in our laboratory at Cambridge. A considerable number of these were caught for us by the keepers, and since it is no doubt easier to catch a feeble bird than a strong one, it may be that they do not fairly represent the average normal bird. It is probable, however, that they do not fall far short, if at all, of this standard, for they were plump and of good weight. They, of course, contained numerous entozoa, as also do the strongest birds which fall to the gun. The rest were hand-reared birds kept in captivity.

The first to attribute grouse disease to a living parasite was, we believe, Cobbold[1] (1873) who drew attention to the presence of the small nematode worm, *Trichostrongylus pergracilis*, often in large numbers in the caeca of grouse which were supposed to have died of the disease. Nineteen years later Klein[2] (1892) investigated the disease, and came to the conclusion that it was "an acute infectious pneumonia" caused by a specific bacillus, which he found in the blood and organs of birds which had succumbed to the disease. But neither of these theories of grouse disease has found general acceptance. Against Cobbold's view is often urged the well established fact that *Trichostrongylus pergracilis* is present practically without exception in the normal wild grouse, and often too in extraordinary numbers, and it has never hitherto been clearly shown to be more numerous in individuals believed to be suffering from grouse disease than in others. Klein's bacillus on the other hand has long been suspected of being no other than *B. coli*, which, as is well known, rapidly invades the tissues after death. To these points we must return when we have recorded our own observations.

[1] *The Grouse Disease*, *The Field* Office, London, 1873.

[2] *The etiology and pathology of Grouse Disease, Fowl Enteritis and some other diseases affecting birds.* 1892. London, Macmillan & Co.

It is necessary at this stage to explain how we came to be associated with the work and what facilities we had for carrying it out.

Grouse disease having been reported in Scotland early in May 1908 we were invited to undertake bacteriological investigations, and accordingly one of us (L. C.) proceeded north to commence preliminary work. Through the kindness of Lord Lovat some rooms at Beaufort Castle were converted into a temporary laboratory, and every effort made by his staff of keepers to procure diseased birds in a living condition. At the same time owners of grouse moors in the neighbourhood were asked to procure living sickly birds if possible. A few days later a move was made to Mr Perrins' moor at Ardross, where Mr Cuthbert, his agent, kindly placed a room in his own house at our disposal for use as a laboratory. The visit terminated after a week as it was necessary to return to Cambridge, but during the time 11 diseased birds and one normal bird were examined together with others picked up dead on the moors. The latter were, of course, useless for bacteriological examination since in the organs of birds which have been dead for some time, whether diseased or healthy, and of birds which have been shot and wounded in the abdomen, *B. coli* and other intestinal bacteria swarm in all the cultures.

A second visit to Scotland was made from August the 25th to September the 1st. During that time eight fresh grouse were examined for bacteria, one being a bird caught when obviously ill on Cawdor moor and received alive.

After this work was continued in Cambridge on normal birds which reached us alive, and on sickly birds caught alive, immediately killed and packed in ice and sent to us from time to time from various moors as occasion offered.

During the visits to Scotland it was, of course, not possible to carry out all the precautionary measures which are described later as having been taken when working in our own laboratories at Cambridge, but the most important of these precautions were observed, such, for example, as plucking the birds before they were brought into the laboratory, the free use of the flame for singeing, and of the actual cautery for destroying any stray particles of feather and for burning the skin through which the incisions were made. In the preliminary experiments also the method of getting at the lungs from behind, which is described later, was adopted; and emulsions of the organs were made between sterilised plates, but the glass frame, in which this was done at Cambridge, had not been adopted at the time of the earlier experiments.

One of the first objects of the experiments was to seek for Klein's bacillus and to compare it with other members of the colon group in the light of the great advances in bacteriology since Klein's observations were made 18 years ago. Our next was to look for characteristic lesions.

The preliminary observations in Scotland showed at once the presence of bacilli of the colon type, which could not be distinguished from Klein's bacillus, in the livers and sometimes in the other organs of diseased grouse, but it soon became evident that these micro-organisms might be present also in the organs of grouse presumably quite normal. No pneumonia was seen in any of the birds examined by us in a perfectly fresh condition, the lungs being always pale pink in colour and free from congestion. In birds picked up dead on the moor it was not always easy to make a definite statement about the lungs as they were often deeply stained and otherwise altered, but in the fresher specimens it was apparent that there was no pneumonia. In the fresh diseased birds the livers were not obviously altered, though in those birds which were picked up dead they often showed more or less of that blackish colour, which has sometimes been described as characteristic of grouse disease, but which is certainly due to post-mortem changes. We had the opportunity of seeing many of these birds through the kindness of Dr E. A. Wilson, who was working at Beaufort at the same time, and it was he who first pointed out to us the entozoal and other parasites of the grouse. He too it was who first pointed out to us that the most notable lesions were in the caeca. The mucous membrane often appeared deeply reddened along the convexities of the longitudinal ridges, and sometimes thickened. To the naked eye, or with the aid of a hand lens, it was plain that considerable pathological change had taken place here, but there was no obvious ulceration. There were always large numbers of strongyli in these caeca. This condition was most advanced in birds which were picked up dead, but it was no post-mortem change, for it was found also in weakly birds which were brought to us alive. There were in these birds also many large tape-worms, *Davainea urogalli*, in the intestine, fragments of which were also rarely found in the caeca. In the birds examined during the spring there were invariably enormous numbers of the slender tape-worm, *Hymenolepis microps*, in the duodenum, and the mucous membrane of this part of the intestine was reddened.

It therefore remained to carefully compare normal and diseased birds (*a*) as to numbers of strongyli, (*b*) liability to contain living

bacilli in their organs, and (*c*) to make a detailed examination of the lesions in the caecal mucous membrane and to see what relation this had to the nematodes on the one hand and the bacilli on the other, and lastly (*d*) to find out whether or no the bacilli exerted any pathogenic action. It seemed possible that the strongyli might be the cause of the changes in the caecal mucous membrane: that these changes might admit the intestinal bacteria to the liver and other organs of the body, and that these together with other pathogenic products abnormally absorbed from the diseased caeca, or possibly the mere interference with absorption caused by that disease, might lead to the death of the birds.

All the diseased birds examined were considerably under weight and wasted. We never came across any instance of a bird dying plump and in good condition unless indeed its death could clearly be attributed to some other cause, such as having flown against a wall.

Methods of making cultures from the organs of birds.

It was recognized from the first that if micro-organisms were present only in small numbers in the organs somewhat large amounts of tissue might have to be used in order to obtain colonies on solid culture media. It was further recognized that the tissue would have to be crushed into a pulp, which could be spread more or less evenly over the surface of the medium, in order that any micro-organisms which might be present should have a chance of coming into contact with it. Moreover, it was clearly seen that in carrying out experiments of this kind the chances of accidental contamination were not inconsiderable. The methods which were first employed under rather difficult conditions in Scotland were later somewhat modified when the investigations were subsequently continued in Cambridge.

The precise conditions under which these experiments were conducted are matters of considerable importance, since upon them depend the reliability of the results which were obtained. We have therefore no hesitation in describing the methods in detail.

1. *Precautions against aerial contamination.*

Previous to beginning an experiment the room was carefully prepared. All dust was removed from the window ledges and elsewhere, and the floor and bench were flooded with a mixture of glycerine and lysol to lay the dust. All the windows and ventilation shafts were

closed during the actual operation of making the cultures. As a further precaution against aerial contamination the tissues were crushed inside a glass frame (Plate I, fig. 1). Two sheets of plate glass, 21 × 8 inches, formed the top and bottom respectively, the former being supported on blocks of wood, which formed the sides. The back was also formed of a sheet of plate glass, and the front was closed by a curtain of linen, which was soaked in lysol and could be partially turned back when required. The joints of the frame were made draught proof by means of rubber tubing. On the floor of the frame another sheet of plate glass, which extended the whole length, but was three inches narrower than the bottom, was placed to form a ledge near the centre of the floor, upon which the plates used for crushing the tissues could be conveniently manipulated, and yet be covered by the roof. The height of the frame from the top to this ledge was three and a half inches.

Before use the frame was washed out with a mixture of glycerine and lysol. In order to estimate the risk of aerial contamination agar plates were exposed on the bench and inside the frame during the whole period of time the cultures were being made.

2. *The preparation of the bird.*

The birds, if living, were killed by decapitation, weighed and immediately plucked in an adjoining room. As far as possible all the larger feathers, except those of the wings, were removed, and the cloaca, if gaping, was plugged with a pledget of cotton wool. The smaller feathers appeared to us to be a particularly dangerous source of contamination, since some might be soiled with faecal matter. Owing to their extreme lightness some of these unless carefully destroyed might float in the air and alight on to the tissues during the manipulations. The smallest feathers are only seen with difficulty, and might easily contaminate pieces of tissue as they were being removed from the body. In order to obviate all chance of contamination from feathers the body of the bird after plucking was held in the flame until all the minute feathers had been completely destroyed.

3. *The method of obtaining portions of the organs.*

A plumber's soldering iron, heated to redness, was freely used to burn the skin through which the incisions for removing pieces of tissue were to be made. The necessary incisions were then made without

delay with instruments sterilised by boiling for at least half-an-hour. A fresh pair of scissors and forceps were used for removing the piece of tissue actually used for cultivation. Cultivations were made from each organ in turn, observing the precautions which have just been described in each case. *Lungs.*—The lungs were approached from the back. After the skin had been thoroughly seared with the iron the muscles under the scapula were transfixed with a knife and the scapula freed by carrying the knife out to its apex; the bone was then turned up and broken. Next two or more ribs were cut through in two places, about half an inch apart, with scissors, and a piece of the lung approximately equal in bulk to a cube one-quarter of an inch in all dimensions was cut out, and quickly transferred to the ground glass plates for disintegration. *Kidneys.*—As the kidneys were approached from the back they were taken immediately after the lungs. A piece of the thin iliac bone, where it bulges outwards, was removed, care being taken not to force the intestines upwards during the process by pressure on the body. The satisfactory removal of portions of the kidneys was often a difficult matter, partly owing to the limited size of the opening which could be conveniently made in the bone, and partly owing to the nerve trunks which traverse the organs and render the extraction of portions difficult. In a few cases the intestine was wounded, but when this accident was perceived at once the attempt to obtain any further cultures from this bird was abandoned. *Liver, Pancreas and Spleen.*—These organs were approached from the front by turning the sternum back after cauterising the whole ventral surface, and especially the lines of the incision. Culture tubes were always sown from the liver, but the pancreas was only examined culturally on a few occasions, and cultures from the spleen were not made when the organ was required for histological purposes. In the grouse the spleen is extremely small, so that even when cultures were made the amount of material employed was considerably less than in the case of other organs. *Blood and Bile.*—Samples of blood were obtained by plunging sterile pipettes through the heart wall after cauterisation. Bile was also obtained in glass pipettes from the gall bladder, but the surface of the latter organ was not cauterised.

4. *The method of crushing the tissues.*

From each organ a piece, at least a quarter of an inch square, was removed by the methods just described, and placed on the surface of a ground glass plate. The plates used were 3 to 4 inches in diameter

and were ground on one side; these were sterilised by boiling and dried separately in the flame. As soon as they were dry the plates were placed in pairs, with their ground surfaces in contact on the glass ledge which has been described in the glass frame; in this situation they cooled rapidly. When a portion of an organ was ready to be ground up the upper plate of a pair was taken up and held in the fingers in such a way that about one-half or one-quarter of it overlapped an equal area of the lower plate. The piece of tissue was then placed between the overlapping areas and crushed. It was not found necessary to use powdered glass or other material to assist disintegration, because the organs of the bird, protected as they are from violence by the comparatively rigid skeleton, are much softer than those of mammals, and are easily reduced to the condition of an emulsion.

5. *The method of making cultures.*

Before starting an experiment a series of sloped agar tubes were labelled, two or three for each organ, and arranged on the bench in the order in which the organs were to be dealt with. As soon as a portion of an organ had been reduced to a pulp a considerable quantity of the pulp was taken up on a sterile platinum wire, bent into a series of loops so as to form a spatula, and spread over the surface of one of the agar tubes. The whole of the material crushed was left on the two tubes. In this way any living organisms that might be present had an opportunity of producing colonies on the surface of the medium. In the case of 9 birds (Nos. 20—28) anaerobic cultures in Buchner tubes were also made from all the organs, but as they did not yield anything more than the ordinary cultures, such cultures were not made in the later experiments.

6. *The examination of cultures.*

The cultures were incubated at 37° C. and examined daily on the first few days, and subsequently at various intervals up to a fortnight. Colonies of *B. coli* or *streptococci* seldom appeared after 24—48 hours' cultivation, except when they grew out of one of the larger masses of tissue on the surface of the tube. The principal result of allowing the cultures to incubate for longer periods was to reveal the presence of moulds and streptothrices, and occasional spore-bearing bacilli and cocci in cultures from the lungs.

The examination of the agar plates, exposed on the bench and within the glass frame during the progress of the experiments, showed that in spite of the long exposure very few colonies grew on them. *B. coli* was never found and moulds and streptothrices were uncommon. The commonest organisms were *S. lutea* and cocci.

7. *The identification of the organisms found.*

All organisms resembling *B. coli* were isolated in pure culture and the characters of their growth on agar, gelatin and potato together with their staining reactions and motility investigated. They were also cultivated up to 14 days in milk and in peptone water tubes containing glucose, lactose, mannite, saccharose, and dulcite.

Altogether 35 lactose fermenting organisms of the colon group were isolated from the organs of the birds and thoroughly investigated. All these organisms had the following characters in common :—Short, Gram negative, non-spore-producing bacilli with rounded ends. Greyish white colonies on agar, gelatin never liquefied. Moist white or cream coloured growth on potato. Permanent acidity and clotting in milk. Most of them produced indol, and the majority showed some motility, especially in peptone water cultures.

Following MacConkey's[1] (1905) classification of the lactose fermenting bacilli, these organisms may be divided, according to their reactions, which are given in the following table, into four groups.

Group	Type	Glucose	Lactose	Saccharose	Dulcite	Mannite	Milk	Indol
I.	*B. acidi lactici* (Hüppe)	A+G	A+G	0	0	A+G	A+C	+
II.	*B. coli communis* (Escherich)	A+G	A+G	0	A+G	A+G	A+C	+
III.	*B. coli communior* (Durham)	A+G	A+G	A+G	A+G	A+G	A+C	+
IV.	*B. lactis aerogenes* (Escherich)	A+G	A+G	A+G	0	A+G	A+C	+

0 = No change. A+G = Acid and gas produced.
A+C = Acid and clot. + = Positive reaction.

Twelve organisms, namely those isolated from the livers of grouse 3, 4, 7, 12, 44, 50, 56 and 60, from the lungs of 15, 50 and 57 and from the blood of grouse 15, belonged to group I; 3, isolated from the livers of grouse 11 and 53 and from the lungs of grouse 1, belonged to group II; 11, isolated from the livers of grouse 1, 6, 13, 16, 46, 51, 54, 58, 59 and 61 and from the lungs of grouse 13, belonged to group III; and 9 isolated

[1] MacConkey, A. (1905). "Lactose-fermenting bacteria in faeces," *Journ. of Hygiene*, v. p. 333.

from the livers of grouse 2, 22, 23, 57 and 62, from the lungs of grouse 22 and 56, and from the spleen and kidney of grouse 22, belonged to group IV.

Organisms belonging to all four groups were cultivated also from the caecal contents on various occasions.

An organism with the same general morphological and cultural characters, but differing in its fermentation reactions, was cultivated from the liver and spleen of grouse 47, and from both lungs, spleen, pancreas and both kidneys of grouse 51. This organism produces acid and gas in media containing glucose, mannite and dulcite, and acidity followed by alkalinity in milk. In media containing lactose and saccharose no change is produced. It corresponds therefore in its cultural characters with the *B. enteritidis* (Gaertner) group.

Many of the other organisms found were similarly investigated except moulds, streptothrices, cocci and spore-bearing bacilli, but in view of their extreme rarity it seems scarcely necessary to give their cultural characters in detail.

The method of counting strongyli in the caeca.

At an early stage in the investigations it began to appear probable that the presence of *B. coli* in the liver and other organs of the grouse was related in some way to the numbers of *Trichostrongylus pergracilis* in the caeca. Up to December 1908 the strongyli were only roughly estimated, but at that time a method of isolating and counting them was devised and found to be practicable, and from that time onwards the strongyli were counted in every case. The method was as follows: The caeca were laid out straight on a board and opened throughout their length, their contents turned out and their mucous membrane scraped. All the material liable to contain strongyli was thus collected. Little by little this was shaken up with water in a large test-tube and poured out drop by drop into a Petri dish containing water. With suitable illumination the strongyli could be clearly seen and picked out with a mounted needle and counted. When the contents of the caeca were drier than usual, and did not readily break up when shaken with water, they were disintegrated by rubbing between the flat surface of a rubber bung and the bottom of a Petri dish. There can be no doubt that, while some strongyli must have escaped notice, this method gave a close approximation to the numbers which were actually present, quite close enough for the purposes of our inquiry. In nearly all cases the worms

in the two caeca were separately counted, usually by different observers. As may be seen, by reference to Table I and Plate II, fig. 3, in all but two birds (56 and 67) approximately equal numbers were present in the two caeca. We thought therefore that in our future investigations a sufficiently accurate estimation of the number of strongyli might be arrived at by counting those present in one caecum and doubling the number found.

TABLE I.

Showing the results of counting the strongyli in the two caeca separately.

Grouse No.	Strongyli		Total	
	One caecum	Other caecum		
52	0	0	0	23 specimens of *Heterakis papillosa*
81	0	0	0	found in one caecum and 10 in
58	54	59	113	the other.
65	89	94	183	
59	108	127	235	1 specimen of *H. papillosa* in each
46	131	128	259	caecum.
55	201	214	415	
63	281	252	533	
64	268	303	571	
57	331	268	599	
62	365	375	730	
67	285	548	833	
68	420	457	877	
66	455	490	945	
56	754	1114	1868	
53	1103	1403	2506	
60	3118	2877	5995	1 specimen of *H. papillosa* in each
61	4769	4793	9562	caecum.

General results of bacteriological examinations of the organs.

In the lungs, moulds and streptothrices were almost constantly found. The fact that they were absent in all but a very few of the tubes sown from other organs indicates that they were really in the birds' lungs during life, and did not get into the tubes as a result of contamination. Further, these results have been confirmed by observations on a number of other animals, both mammals and birds.

The other organs and blood were in the immense majority of cases free from cultivatable micro-organisms, except when *B. coli* was present. Occasionally a single colony of some microbe would appear, perhaps a spore-bearing bacillus like *B. subtilis*, or *Sarcina lutea*, or rarely a mould.

On several occasions diphtheroid segmented bacilli were found. That these were sometimes accidental contaminations seems very probable, and in any case their numbers were so few as to be of little practical importance. Nevertheless, it may be that some were really in the living tissues during life, and this seems more probable in the case of the segmented bacilli, which were sometimes found in cultures from the blood, which are less liable to contamination than those from the solid organs, as well as elsewhere. Moreover in one case (grouse 37) they were also cultivated from the contents of the intestinal canal, but were only rarely met with on the exposed agar plates.

The whole question of the presence of bacteria in the living organs is in the course of investigation by us, and we need not dwell further on the matter here, except in so far as *B. coli* is concerned.

Note on the alimentary canal of the normal grouse[1].

Beyond the oesophagus, crop and gizzard the alimentary canal consists of the duodenum, intestine, paired caeca and rectum (Plate I, fig. 2).

The duodenum, a thin-walled light-coloured tube, four to seven inches long, on which the vessels are clearly seen, forms a U-shaped loop, of which the limbs lie in close contact with one another, the angular space on the ventral side being occupied by the pancreas. Next follows the intestine, a thicker walled tube of grey colour some 20 to 34 inches in length, and half an inch in diameter. From the junction of the intestine and rectum arise the paired caeca. Each caecum consists of a short narrow portion with small lumen next the intestine, and a long wider portion between one and two feet, or even more, in length, and about one-third of an inch in diameter. At its distal end it tapers rather suddenly to a point. Its walls are thinner than those of the intestine and are marked by about nine longitudinal whitish lines. On opening the caecum well marked longitudinal ridges are seen, corresponding to the lines just described. Each ridge shows alternate thicker and thinner portions. Occasionally one of the ridges may be seen to die away or fuse with its neighbour. They occur throughout the whole length of the caecum.

On examination under a Zeiss binocular microscope (× 8—33) the mucous membrane (of a grouse (No. 81) in which no strongyli are

[1] The measurements of the various parts of the alimentary canal vary greatly in different birds.

present), after gentle washing in a stream of water, is seen to be regularly beset with small villi of uniform size, arranged closely together on the ridges, but more widely separated in the depressions, where they seem to be less well developed. They often appear club-shaped, more especially on the ridges, where their flattened terminations, lying closely together at a uniform level, give the surface a somewhat smooth and tessellated appearance (Plate IV, fig. 11). In birds (e.g. No. 69) caught on the moor and apparently normal but infected with strongyli both the ridges and the villi are much larger (Plate IV, fig. 12).

The rectum is a thick walled tube of greyish white colour, about four inches in length.

The contents of the gut vary much in different parts. The duodenum usually contains nothing but a white slimy mucus. The intestine contains coarsely divided particles of food and occasional stones from the gizzard. The contents of the caeca present a marked contrast to those of the intestine, consisting of a brownish or greenish pasty mass of finely divided material. The rectum contains usually only the coarser particles of food which have never passed into the caeca.

Pathological changes in the alimentary canal.

Duodenum. At certain seasons of the year the duodenum of every wild bird examined was packed with the long tape worm *Hymenolepis microps.* They were particularly numerous from March to May and towards the end of August. Under these circumstances the contents appear to consist wholly of tenacious mucus, until shaken up in alcohol, when the worm becomes visible for the first time. No obvious pathological changes, except some reddening, were seen. *Trichosoma longicolle* was occasionally found in small numbers.

Intestine. The lower half of the intestine was often found distended with tangled masses of the large tape-worm, *Davainea urogalli*; they bear a less definite relation to season than does *Hymenolepis.* Portions of these worms are sometimes found bile stained. No pathological changes were noticed.

Caeca. The appearance of the caecum as seen from without varied; in some cases there were no obvious changes; in some cases the caeca appeared to be somewhat dilated, in others they appeared mottled with lighter coloured patches. The contents, the main portion of which was semi-fluid, often contained—especially near the proximal ends—dry masses, which were very adherent to the mucous membrane, and which

corresponded to the whitish patches seen from the exterior. Whenever one of these masses was pealed off numerous strongyli could be seen stretched between the mass and the mucous membrane, obviously adherent to both. The dry attached condition of these masses strongly suggested that they represented material which had long been retained in the gut.

The small nematode, *Trichostrongylus pergracilis*, was often present in enormous numbers, occasionally amounting to thousands. They were particularly numerous towards the proximal ends of the caeca, especially in the dry masses just described. Except in one instance we never failed to find strongyli in wild grouse, and they were always present in large numbers in birds suffering from grouse disease. The numbers present in wild grouse did not appear to depend in any way upon the time of year. Portions of *Davainea* were on rare occasions seen in the caeca.

After washing in a gentle stream of water the mucous membrane frequently appeared reddened, especially in birds which were picked up dead on the moor. The reddening was present in many, but not in all, of the birds badly infested with strongyli which were examined in a perfectly fresh condition. It was thought that this might possibly have been a post-mortem change, and some normal birds were kept after death for a few days before examination to see if the redness would appear in them, but it was not seen. When examined under a Zeiss binocular microscope (× 8—33) the ridges were found to be thickened, especially in patches, to which the dry masses already referred to were found adherent (Plate IV, fig. 13). The villi were very irregular in all situations, being in places greatly hypertrophied and club-shaped both in the depressions and on the ridges, and in other places atrophied, particularly on the thickenings just mentioned. In many cases the villi on the ridges were embedded in some cementing material, which in microscopic sections appeared to be composed of a mixture of mucous and granular debris, which could not be removed by gentle washing. Even after free washing numerous strongyli could be seen adherent to the mucous membrane, and frequently penetrating between the villi (Plate V, fig. 17). In some of the worst cases the ridges are so deformed as to resemble masses of coral, with smooth but irregular surfaces, on which the individual villi are frequently indistinguishable, and with cave-like depressions between them from which one or more strongyli can be seen protruding (Plate IV, fig. 14). These appearances, we believe, are due to the matting together of the villi and

sometimes of the neighbouring ridges by the cementing material described above.

Histological changes in the caeca.

With the small amount of material at our disposal[1] it was impossible to follow out in detail the various changes which occur in the caeca, and we therefore confine ourselves to comparing the condition found in severely affected birds with that found in normal birds. In the investigation of the histological changes we had the advantage of the expert opinion of Mr T. S. P. Strangeways, Huddersfield Lecturer in Special Pathology, Cambridge, to whom we are greatly indebted.

Sections of the caecum of the normal bird (No. 81) without strongyli show the following structures.

There is under the peritoneum a well marked muscular coat, and within this delicate areolar tissue supporting a layer of well formed connective tissue on which the mucous membrane rests. At intervals the connective tissue layer projects towards the lumen of the gut forming the central core of the ridges which have been described. At their bases these prolongations appear bifurcated, and the spaces between the bifurcations are filled with fat and some large blood vessels. Both the ridges and the depressions between them are covered with villi of fairly uniform length, which consist of a central core of vessels surrounded by a small quantity of delicate sub-epithelial connective tissue, together with a few lymphoid cells, covered with a single layer of columnar epithelium. Here and there in the depressions may be seen sections of lymphoid follicles covered with villi. The contents lying in the lumen of the gut consist of a mass of granular material and mucus (Plate II, figs. 5 and 6).

Sections of the caecum of an apparently healthy grouse (No. 69) with many strongyli (1460) caught on the moor differ in certain respects (Plate III, fig. 7). The muscular walls contain distinct bands of wavy fibrous tissue. The quantity of fibrous tissue in the cores of the ridges seems to be increased; but fat is still present in the bifurcations. The ridges are large and the villi are markedly increased in size, especially those situated near the free margins of the ridges. In the latter wavy bands of fibrous tissue may be seen; and lymphoid cells are found in considerable numbers within all the villi. The epithelium appears hypertrophied, but is not markedly irregular except over the villi on the free

[1] In 26 specimens the contents of both caeca were used for counting the strongyli; and 14 specimens arrived dead and therefore useless for minute histological examination.

margins of the ridges. Worms are uncommon except in certain situations in the depressions, where they seem to be entangled in what appears to be dry, concentrated gut contents. No lymphoid follicles can be seen.

In a diseased grouse (No. 6), in which the macroscopic changes are well marked, the following condition is found. The muscular wall contains well marked strands of fibrous tissue. The fat at the bases of the ridges has completely disappeared, and the vessels show considerable thickening of their walls. The connective tissue in the cores of the ridges is also greatly increased in amount and in density and the vessels dilated. The sub-epithelial connective tissue of the villi is also increased in amount and the vessels in it dilated, and probably increased in number, and in some cases full of blood. The connective tissue is in most places loose and contains large numbers of cells, probably inflammatory in origin, and in some places, especially near the free ends of the villi and in the neighbourhood of the worms, shows fibroid change. The epithelium is proliferated and thrown into folds (Plate III, fig. 8).

In a grouse (No. 15) badly infected with strongyli and showing well marked macroscopic lesions all the changes just described are more evident. Much fibrous tissue is present in the muscular coat, and the walls of the vessels are very markedly thickened. The villi appear increased in size, and their connective tissue is more dense, and contains a considerable amount of fibrous tissue, replacing the more delicate connective tissue. In this tissue a large number of the nuclei are clearly those of newly formed fibrous tissue, being elongated and spindle-shaped, though round cells are still present in considerable numbers. Nuclei of the former type are now found in all situations, and are not limited to the cores of the ridges as in the case of specimens from normal birds. The epithelium shows great proliferative changes and is thrown into irregular folds. In all specimens from diseased birds the lymphoid follicles are indistinguishable (Plate III, figs. 9 and 10).

In fact, the general condition shows evidence of a chronic inflammation leading to fibrosis. Large quantities of mucus are present in the intestinal contents, and the villi appear to be united together with this material, which penetrates to the deepest parts of the crypts between the villi. Everywhere strongyli are present, and their relationship to the structures composing the wall of the organ is of special interest (Plate III, fig. 10). They are found in large numbers both in the lumen and between the villi, in some instances having penetrated to the deepest portions of the crypts. In such cases the epithelium covering the portions of the villi adjacent to the worms is greatly altered, and a

marked increase of fibrous tissue in the underlying connective tissue is frequently observed (Plate V, fig. 18). In some instances the epithelium has completely disappeared all round the worm so that the latter is seen surrounded by a ring of dense fibrous tissue (Plate IV, fig. 16). Occasionally a worm is found lying between the epithelium and the matrix of the villus, which usually shows fibroid change in the neighbourhood (Plate IV, fig. 15).

There can be little doubt therefore that the presence of the worms in such situations leads to chronic inflammatory changes and fibrosis.

The relation of B. coli in the organs to strongyli in the caeca.

As has already been stated it began to appear probable early in the course of the work that the presence of *B. coli* in the liver and other organs was related in some way to the numbers of strongyli in the caeca. In several birds which had been raised in captivity in Scotland and subsequently kept in Surrey no strongyli could be found, even after a careful search, and in their organs there were no bacilli of the colon type (one exception). On the other hand, in the organs of grouse with very large numbers of strongyli *B. coli* was constantly present, either in the liver or in some other organ. In other grouse with fewer strongyli *B. coli* was present in some and appeared to be absent in others.

The results obtained previous to the adoption of the counting method are shown in Table II, p. 19, in which the birds are arranged in three classes.

In the birds of class I *B. coli* was found once only in the organs, and then in a grouse with very numerous tape worms in the duodenum and intestine, and numerous portions of *Davainea* in the caeca, a thing very rarely observed and probably indicating some abnormal condition. *B. coli* was found in the liver of one of six birds belonging to class II. Amongst the birds belonging to class III *B. coli* was constantly found in the liver (one exception). In five instances they were cultivated from one of the other organs also.

Since the counting of the strongyli was systematically undertaken 23 presumably healthy birds have been examined. The results confirmed the opinions previously arrived at. In this series four birds had less than 100 strongyli, and *B. coli* was not found in their organs. Fifteen had strongyli varying in number between 100 and 1000, and *B. coli* was cultivated from the organs of some (8) and not from those of

TABLE II.

Showing the results of cultures previous to the adoption of the method of counting strongyli.

	Grouse No.	Intestinal worms			Cultures from the organs [1]			
		Hymenolepis	*Davainea*	*Strongylus*	Liver	Lungs	Spleen	Kidney
Class I. No strongyli. No *B. coli*.	5.	0	0	–	0	0	0	–
	18.	0	0	0	0	0	0	0
	19.	0	0	0	0	0	0	0
	21.	0	0	0	0	0	0	0
	23.	Numerous	Numerous	0	*B. coli*	0	*B. coli*	0*
	25.	0	0	0	0	0	0	0
	26.	0	One	0	0	0	0	0
Class II. [2]Few strongyli. *B. coli* inconstant.	16.	0	0	Few	–	0	–	–
	17.	Few	One	Eggs only	0	0	0	0
	30.	Few	Numerous	Few	0	0	0	0
	33.	Moderate	Numerous	Few	*B. coli*	0	0	0
	34.	Few	Moderate	Few	0	0	0	0
	40.	0	0	Few	0	0	0	–
Class III. Many strongyli. *B. coli* constant.	1.	Numerous	–	Numerous	*B. coli*	*B. coli*	–	–
	2.	Numerous	Numerous	Numerous	*B. coli*	0	–	–
	3.	Numerous	Few	Numerous	*B. coli*	0	–	–
	4.	Numerous	Numerous	Numerous	*B. coli*	0	–	–
	6.	Numerous	Numerous	Numerous	*B. coli*	0	–	–
	11.	Numerous	Numerous	Numerous	*B. coli*	0	–	–
	12.	Numerous	Numerous	Numerous	*B. coli*	0	–	–
	13.	Numerous	0	Numerous	*B. coli*	*B. coli*	–	–
	14.	Numerous	0	Numerous	*B. coli*	0	–	–
	15.	Numerous	Numerous	Numerous	*B. coli*	*B. coli*	–	–
	22.	0	Two	Numerous	*B. coli*	0	0	*B. coli*
	28.	0	0	Numerous	*B. coli*	0	0	0
	29.	0	0	Numerous	*B. coli*	0	0	0
	31.	Numerous	Few	Numerous	0	0	*B. coli*	0
	32.	Moderate	Moderate	Numerous	*B. coli*	0	0	0
	35.	Numerous	Numerous	Numerous	*B. coli*	0	0	0

* Numerous portions of *Davainea* in the caeca.

[1] In this and the following Table 0 indicates that no organisms of the *B. coli* type were present in the cultures, and – that cultures were not made.

[2] In the birds of Class II the strongyli were noted as few, but subsequent experience with counting methods showed that what appeared few to an ordinary examination might sometimes turn out to be 100 or more when counted.

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others (7). In four birds in which very large numbers of strongyli, *i.e.* over 1000, were counted *B. coli* was found in the livers of all. The results of these observations are given in the following table.

TABLE III.

Showing the results of cultures after the adoption of the method of counting strongyli.

Grouse No.	Number of strongyli	Cultures from organs			
		Liver	Lungs	Spleen	Kidneys
52	0	0	0	0	0
81	0	0	0	0	0
48	32	0	0	0	0
43	45	0	0	0	0
58	113	0	0	0	0
59	235	*B. coli*	0	0	0
46	259	*B. coli*	0	0	0
50	290	*B. coli*	*B. coli*	*B. coli*	0
49	330	0	0	0	0
47	344	*B. ent.*	0	*B. ent.*	0
55	415	0	0	0	0
63	533	0	0	0	0
51	540	*B. coli*	*B. ent.*	*B. ent.*	*B. ent.*
64	571	0	0	0	0
57	599	*B. coli*	0	0	0
62	730	*B. coli*	0	0	0
67	833	0	0	0	0
44	871	*B. coli*	0	0	0
66	945	0	0	0	0
54	1645	*B. coli*	0	*B. coli*	0
56	1868	*B. coli*	*B. coli*	0	–
60	5995	*B. coli*	0	0	0
61	9562	*B. coli*	0	0	0

B. ent. = *B. enteritidis.*

The points which come out clearly from these two tables are:—(1) that when strongyli are absent from the caeca or are present only in small numbers (less that 100) intestinal bacteria, especially *B. coli*, are not present in the liver or other organs of the grouse (11 grouse—1 exception); (2) that when relatively few strongyli are present (100—1000) *B. coli* may or may not be present in the organs (21 grouse); and (3) that when enormous numbers (over 1000) are present *B. coli* has invariably been found in the liver or other organs (20 grouse).

It has not been found possible to estimate the numbers of living *B. coli* present from the number of colonies which grew on the tubes.

Sometimes of course we were able to make a rough guess. In some control birds both diseased and healthy, which were examined some considerable time after death, the innumerable number of colonies on the tubes showed that *B. coli* was at that time swarming in the tissues. But with living birds, even when very large numbers of strongyli were present, the colonies of *B. coli* were few in number, one or two to ten or a dozen, and rarely more than thirty.

It is not claimed, of course, that the number of living *B. coli*, in the liver for example, is exactly proportional to the number of strongyli. With as few strongyli as 235, *B. coli* has been found (one colony in one tube), and again with as many as 945, *B. coli* has been absent. Doubtless other conditions which affect the health of the bird influence the permeability of the intestinal wall to the contained bacteria.

The numbers of strongyli present in healthy and diseased birds.

We have already shown that strongyli are almost constantly present in the caeca of wild grouse believed to be perfectly normal, and certainly of fair weight and in good general condition. In a few so-called healthy birds they may be present literally in thousands. We were informed by Dr Wilson that strongyli are more numerous in diseased than in healthy birds; and we have ourselves examined a number of diseased birds brought in dead, and useless for cultural purposes, and collected the worms from them.

Table IV shows that the number of strongyli present in diseased birds, though varying considerably, is greatly in excess of that found in the great majority of normal birds. In a small minority of the presumably healthy birds the numbers were as large as those found in many of the diseased birds. It is, of course, impossible to be certain that these exceptional birds were not really suffering from the early stages of grouse disease. The two (60, 61) with the largest numbers came from a moor on which grouse disease was prevalent at the time.

The presence in diseased birds of strongyli in numbers far in excess of those found in normal birds does not of course prove that they were the cause of the disease, because it is conceivable that they may have multiplied as a consequence of the disease. Nevertheless, taken in conjunction with the changes previously described in the mucous membrane of the caecum and the relation of the worms thereto, it is exceedingly probable that the worms are really the cause of the disease.

TABLE IV.

Showing the relative number of strongyli in healthy birds and those believed to be suffering from grouse disease.

Birds received alive, apparently in good health, or sent as average specimens of normal grouse		Diseased birds picked up dead		
Grouse No.	Number of strongyli	Grouse No.	Number of strongyli	
81	0	53	2506	
52	0	79	2556	(1278 in one caecum)
48	32	74	3114	(1557 ,,)
43	45	78	3340	(1670 ,,)
58	113	74 (*a*)	3406	(1703 ,,)
59	235	80	3840	(1920 ,,)
46	259	75	4352	(2176 ,,)
50	290	39	6230	
49	330	71	7058	(3529 in one caecum)
47	344	73	7484	(3742 ,,)
55	415	77	8800	(4400 ,,)
63	533	72	10266	(5133 ,,)
51	540	76	18332	(9166 ,,)
64	571			
57	599			
62	730			
69	730*			
67	833			
44	871			
66	945			
54	1645			
56	1868			
70	2524*			
60	5995†			
61	9562†			

* One caecum only counted and the numbers doubled.

† These birds came from the same moor.

The relation of B. coli in the organs to tape-worms in the intestine.

The question arises whether the tape-worms, often present in enormous numbers (Plate V, figs. 19, 20) in the gut of the grouse, act like the strongylus and increase the permeability of the intestinal wall to bacteria. Tables V and VI show that there is little or no relation between the presence of tape-worms in the gut and *B. coli* in the organs. Numerous tape-worms of either kind, *Hymenolepis* and *Davainea*, might be present without any *B. coli* appearing in cultures from the liver; and on the other hand, *B. coli* might be present in the liver and yet one or other or both of the tape-worms might be absent.

TABLE V.

Showing that the presence of Hymenolepis in the duodenum is not related to the presence of B. coli in the liver.

Grouse No.	*Hymenolepis*	Cultures from the organs			
		Liver	Lungs	Spleen	Kidneys
52	0	0	0	0	0
81	0	0	0	0	0
48	0	0	0	0	0
43	0	0	0	0	0
49	0	0	0	0	0
58	0	0	0	0	0
55	0	0	0	0	0
59	0	*B. coli*	0	0	0
46	0	*B. coli*	0	0	0
50	0	*B. coli*	*B. coli*	*B. coli*	0
47	0	*B. ent.*	0	*B. ent.*	0
51	0	*B. coli*	*B. ent.*	*B. ent.*	*B. ent.*
57	0	*B. coli*	0	0	0
62	0	*B. coli*	0	0	0
44	0	*B. coli*	0	0	0
56	0	*B. coli*	*B. coli*	0	0
54	0	*B. coli*	0	*B. coli*	0
60	Few	*B. coli*	0	0	0
63	Numerous	0	0	0	0
64	Numerous	0	0	0	0
67	Numerous	0	0	0	0
66	Numerous	0	0	0	0
61	Numerous	*B. coli*	0	0	0

B. ent. = *B. enteritidis.*

The prevalence of Hymenolepis and Davainea in normal and diseased birds.

It is not without significance that *Hymenolepis*, so numerous from the spring to the autumn months, during which the greatest mortality takes place, is scarce or absent during the winter, when the disease is quiescent. The enormous numbers which both these worms may attain is almost unbelievable by one who has not seen them (Plate V, figs. 19, 20). *Davainea* seems to be present in the intestine throughout the year. On the other hand, we have not observed any gross lesions in the neighbouring mucous membrane even in the worst cases of infection with either of these worms, nor have they, as has already been shown, any

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relation to the presence of living intestinal bacteria in the tissues. We therefore are not inclined to believe that they play any part, except perhaps a secondary one, in the causation of grouse disease.

TABLE VI.

Showing that the presence of Davainea in the intestine is not related to the presence of B. coli in the liver.

Grouse No.	*Davainea*	Cultures from the organs			
		Liver	Lungs	Spleen	Kidneys
52	0	0	0	0	0
81	0	0	0	0	0
48	0	0	0	0	0
58	0	0	0	0	0
59	0	*B. coli*	0	0	0
46	0	*B. coli*	0	0	0
50	0	*B. coli*	*B. coli*	*B. coli*	0
63	0	0	0	0	0
51	0	*B. coli*	*B. ent.*	*B. ent.*	*B. ent.*
62	0	*B. coli*	0	0	0
44	0	*B. coli*	0	0	0
43	One	0	0	0	0
66	One	0	0	0	0
56	Five	*B. coli*	*B. coli*	0	0
54	Six	*B. coli*	0	*B. coli*	0
47	Seven	*B. ent.*	0	*B. ent.*	0
55	Seven	0	0	0	0
57	Eleven	*B. coli*	0	0	0
67	Moderate	0	0	0	0
49	Moderate	0	0	0	0
64	Numerous	0	0	0	0
60	Numerous	*B. coli*	0	0	0
61	Numerous	*B. coli*	0	0	0

B. ent. = *B. enteritidis.*

In the above tables the birds are arranged in order, according to the numbers of tapeworms present. When two or more birds had the same number of these worms they are arranged according to the number of strongyli.

The relation of B. coli in the liver to coccidiosis of the intestine.

Three grouse chicks, reared at Frimley and experimentally fed on coccidia by Dr Wilson, were examined by cultures for the presence of intestinal organisms in their organs.

The first (B. 15. Hatched 28. VI. 09. Faeces examined and no spores of coccidia found. Fed twice on 9. VII. and 17. VII with faeces

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from other birds containing spores of coccidia. Killed 6. VIII.) was very ill and extremely emaciated when received, and was killed and examined immediately. An organism of the *B. enteritidis* type was found in the liver, but not in the other organs. No worms of any kind were found in the intestine or caeca. Sections of the gut examined by Dr Fantham showed numerous coccidia in all stages of multiplication in the cells. The second chick (B. 2. Hatched at the same time and treated in the same way) was also ill when received, and was killed and examined immediately. In this case a few colonies of *B. coli* were obtained from the liver cultures. No worms were found, and the condition of the intestine was the same as in the first chick. A third older chick (4 months) which had been fed on coccidia three weeks previously was also killed and examined. A few streptothrices developed on the cultures from the lungs, but those from the other organs remained sterile. Neither worms nor coccidia were found in the intestine or caeca.

These observations seem to indicate that intestinal coccidiosis may so injure the gut that bacteria are allowed to pass into the circulation. This conclusion is supported by eight observations on young rabbits suffering from naturally acquired coccidiosis of the intestine, the results of which are given in the following table.

TABLE VII.

Showing the results of cultures from the organs of young rabbits suffering from coccidiosis.

Rabbit No.	Coccidiosis		Cultures from the organs							
	Intestine	Liver	Liver	Spleen	Lungs	Kidneys	Blood	Bile	Mesenteric gland	
1.	No lesions	Well marked	0	0	0	0	0	0	*B. coli.*	No nematodes.
2.	,,	Excessive	0	0	0	0	–	–	,,	,,
3.	,,	Well marked	0	0	0	0	–	–	,,	
4.	Trace only	Few small spots	0	0	0	0	0	–	,,	Many nematodes.
5.	,,	,,	0	0	0	0	0	–	,,	,, (233).
6.	Well marked	,,	*B. coli*	0	0	0	–	–	,,	,, (246).
7.	,,	One spot	*B. enteritidis*	*B. enteritidis*		0	0	–	*B. coli* *B. enteritidis.*	No nematodes.
8.	,,	Well marked	*B. coli*	0	0	0	0	–	*B. coli.*	

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The mesenteric glands yielded intestinal bacteria in all cases. The cultures from the other organs, including the liver, yielded no intestinal bacteria when the small intestine was normal, or showed merely a trace of coccidiosis. On the other hand, when the small intestine showed well marked coccidiosis *B. coli* or *B. enteritidis* was always present in the liver, and sometimes in the other organs. The existence of coccidiosis of the liver bore no relation to the presence of *B. coli* in that organ, even the affected bile ducts being sterile. The presence of the nematode, *Oxyuris ambigua* (in moderate numbers) did not appear to have any influence on the passage of bacteria from the intestine into the blood vessels.

The significance of B. coli in the organs.

We have shown that *B. coli* is constantly present in the organs of birds whose caeca contain large numbers of strongyli, and we have shown that the latter are present in far larger numbers in diseased than in healthy birds. It may therefore be assumed that *B. coli*, while not invariably absent from the organs of the healthy bird, is constantly present in those of diseased birds. The small numbers of colonies of *B. coli* cultivated from the tissues of diseased grouse indicate that these bacteria do not multiply in the tissues. We therefore do not suggest that grouse disease is essentially an infection with these bacteria. But the number of colonies which appear on our tubes does not allow us to estimate even approximately the numbers of bacilli which enter the tissues and get killed. The products of these bacilli, if really numerous, doubtless exert some amount of harmful influence, but how much we are not at present in a position to say. No bacilli could with certainty be identified either in sections or in smears of the livers of diseased or healthy birds. Many of the cells were filled with large numbers of iron-containing granules, which seemed to be more numerous in diseased than in healthy birds. But the fact that we have not found either by macroscopic or microscopic examination any important lesions in the livers which have yielded cultures inclines us to think that the bacilli play only a secondary part in the causation of death.

Seasonal prevalence of the principal grouse entozoa.

Though this subject is being very fully dealt with by other workers for the Grouse Disease Inquiry we feel that it is desirable, in view of the statements we have made, to record our own observations in tabular

TABLE IX, *Summary of all observations.*

Trichostrongylus pergracilis—In Grouse Nos. 1—41 the strongyli were not counted, and only portions of the cæcal contents were examined.

In Grouse Nos. 43—68 (except 45) the strongyli were counted in both cæca separately (see p. 12).

In Grouse Nos. 69—81 (indicated * in Table) the strongyli were counted in one cæcum and the number found doubled.

Number	Date	Locality and History	Sex	Wt. in ozs.	Intestinal worms			Cultures from organs Liver
					Hymenolepis	*Davainea*	*Strongylus*	
1	5. v. 1908	Inverness. Caught unable to fly	♂	16	Numerous	–	Numerous	*B. coli* (12)
2	6. v. 08	,, ,, ,,	♂	16	,,	Numerous	,,	*B. coli* (several)
3	,,	,, ,, ,,	♂	—	,,	Few	,,	*B. coli* (12)
4	,,	,, ,, ,,	♂	20	,,	Numerous	,,	*B. coli* (few)
5	7. v. 08	Normal hand reared bird. Frimley	—	15	0	0	—	0
6	,,	Inverness. Caught unable to fly	♂	16	Numerous	Numerous	Numerous	*B. coli* (several)
11	9. v. 08	,, ,, ,,	♂	17	,,	,,	,,	*B. coli* ,,
12	,,	,, ,, ,,	♀	—	,,	,,	,,	*B. coli* (few)
13	,,	,, ,, ,,	♀	18	,,	0	,,	*B. coli* (1)
14	,,	,, ,, ,,	♂	16	,,	0	,,	*B. coli* (1) *st.c*
15	,,	,, ,, ,,	♂	23	,,	Numerous	,,	*B. coli* (several)
16	2. vi. 08	Normal. Frimley	♂	15	0	0	Few	–
17	10. vii. 08	,, ,, ...	♂	14	Few	1	Eggs only	*sb*
18	,,	,, ,, ...	♂	21	0	0	0	*sb*
19	24. vii. 08	,, ,, ...	♀	23	0	0	0	0
20	28. vii. 08	,, ,, ...	—	15	0	0	Few	–
21	5. viii. 08	,, ,, ...	♀	15	0	0	0	0
22	10. viii. 08	,, ,, ...	♀	—	0	2	Numerous	*B. coli* (1)
23	15. viii. 08	Inverness. Apparently healthy	♂	13	Numerous	Numerous	0	*B. coli* (4)
25	18. viii. 08	Normal. Frimley	♂	15	0	0	0	0
26	,,	,, ,, ...	♀	13	0	1	0	*S. lutea* (1)
28	—	Inverness	—	—	0	0	Numerous	*B. coli* (few)
29	27. viii. 08	,, Caught unable to fly ...	—	15	0	0	,,	*B. coli* (1)
30	26. viii. 08	,, Apparently healthy ...	♀	16	Few	Numerous	Few	0
31	27. viii. 08	,, ,, ,,	♀	20	Numerous	Few	Numerous	0
32	28. viii. 08	,, Healthy, caught on moor	♀	16	Moderate	Moderate	Moderate	*B. coli* (2)
33	,,	,, ,, ,, ,,	♂	15	Numerous	Numerous	Few	*B. coli* (few)
34	29. viii. 08	,, ,, ,, ,,	♂	18	Few	Moderate	,,	0
35	,,	Nairn. Caught on moor. Ill ...	♀	17	Numerous	Numerous	Numerous	*B. coli* (several)
36	3. ix. 08	Normal bird. Examined 4 days after death	♀	—	0	0	0	–
37	14. ix. 08	Normal bird. Found dead. Frimley	♀	11	0	0	0	–
38	23. x. 08	Normal bird. Died of pericarditis	♂	20	0	Numerous	Numerous	*st.c* (1)
39	24. x. 08	Lancashire. Caught unable to fly	♂	18	Few	,,	6230	–

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form. Though unfortunately we seldom had the opportunity of making observations on diseased and healthy birds at the same time the table shows that *Hymenolepis microps* occurred in large numbers in the great majority of birds examined from the middle of March to the end of May. Very few were present in the 13 birds examined between the beginning of June and the last week in August. During the last few days in August they were met with in moderate numbers in all the birds examined. From the beginning of September to the beginning of February they were absent from all the birds examined with one exception[1]. The relation to season is much less marked in the case of *Davainea urogalli*, though it occurred in the greatest numbers at the same seasons as *Hymenolepis*.

With regard to *Trichostrongylus pergracilis* it is difficult to come to any definite conclusion as to its seasonal prevalence from our own observations, conducted as they were on diseased birds at one time of year, and on healthy, often hand-reared, birds at another; but it is clear that they do not disappear at any season.

Summary.

The causes of death of the grouse are, of course, various. We ourselves have seen pleuropneumonia (in a bird long kept in captivity in Cambridge), pericarditis, necrotic patches in the liver, an obscure chronic disease of the peritoneum, and septic infection from a gangrenous fracture of the wing. On the other hand the great majority of birds, either picked up dead on the moor, or caught by keepers when weak and unable to fly, have been found to be all more or less in the same condition; they were wasted, badly infested with *Trichostrongylus pergracilis*, and often also with *Davainea urogalli* or *Hymenolepis microps*, or with both. More or less pathological change was seen in the caeca; the mucous membrane was often reddened, and under the binocular microscope considerable changes were seen, though we did not observe gross ulceration. Sections examined under the higher powers showed serious chronic inflammatory changes particularly in the immediate neighbourhood of the worms.

[1] Our observations have been made on a small number of birds, but Dr E. A. Wilson, who will shortly publish statistics based on the examination of a very large number of birds during several years, informs us that *Davainea* is abundant throughout the year, and that *Hymenolepis* is abundant from May to October. The numbers of the latter gradually diminish from November to February, but rise suddenly in the early part of March.

Birds showing these changes we take to be representative of the chronic form of grouse disease. Whether there be also an acute epizootic disease among grouse we cannot tell. We can only say that, so far as our experience goes, we have not seen it. We have never seen pneumonia in the wild bird, and we have never seen any birds picked up dead when plump and in good condition without finding evidence that they had died of injury.

We have therefore to discuss the causes of death in the chronic wasting disease, which is observed among grouse fairly regularly in the Spring and to a lesser extent in the Autumn, and it is to this we refer when we speak of "grouse disease."

First we must consider the gross intestinal parasites which occur in such remarkable numbers in the grouse. The tape-worm *Hymenolepis microps* alone shows any relation in its seasonal prevalence to grouse disease. This worm, according to our experience, is undoubtedly very numerous in the Spring and Autumn, the seasons when grouse disease is most frequently observed, and practically disappears from the bird during the winter months. On the other hand it has not appeared to be more numerous in diseased than in healthy birds. *Davainea* not infrequently occurs in such enormous masses as to distend the gut.

These tape-worms have not been found associated with any constant or serious lesions. *Davainea* appears to us to be the less objectionable. *Hymenolepis*, whose seasonal prevalence more closely agrees with that of grouse disease, seems to us more likely to be harmful. The large masses in which it often exists in the narrow duodenum appear not unlikely to interfere mechanically with the free passage of food material. Both worms probably make a considerable demand for their own sustenance, even if they do not exert a more serious injurious influence.

The case against the nematode, *Trichostrongylus pergracilis*, is much clearer, for though it is seldom entirely absent from normal birds, nevertheless, definite lesions in the caecum are often associated with its presence in large numbers. It probably, however, does little harm if not too numerous. With regard to the presence of this parasite in large numbers in some of the birds caught on the moor, and supposed to be normal birds, it must be remembered that strong wild grouse are difficult to catch, and that some at least of the methods of capturing grouse alive seem calculated to catch the weakest birds rather than the stronger ones. On the other hand we have counted the strongyli in a number of "normal" and diseased birds, and have found, on the whole, a great difference between the two classes; very large numbers

being always found in the diseased birds, much larger indeed than those found in all but the exceptional members of the healthy class; and these, for reasons just stated, may perhaps be not normal at all but suffering from the early stages of grouse disease.

These nematodes, in birds picked up dead or brought to us by the keepers as suffering from grouse disease, are, so far as our experience goes, almost always associated with grave changes in the mucous membrane of the caecum; and concurrently with these changes intestinal bacteria, particularly those belonging to the *B. coli* group, find their way into the liver, or even into the other organs. We have determined by actual worm counts and cultures that *B. coli* is always absent from the liver (in birds examined immediately after death) when there are no strongyli (hand-reared birds) or only very few (not exceeding 100 in number). When more than 100 but less than 1000 are found then *B. coli* is sometimes present in and sometimes absent from the organs, but when the numbers of strongyli exceed 1000 then *B. coli* is always present in the liver, and occasionally in the other organs.

We have not been able to satisfy ourselves that the bacilli which find their way into the organs do much harm. Some harm no doubt they do, but how much we cannot say. Microscopic examination has not revealed any profound changes in these livers. The numbers in which these bacteria penetrate into the organs is difficult to estimate because, doubtless, they soon get killed in the living tissues, so that the numbers of colonies cultivated must bear only a small proportion to the total number of bacteria which have entered the fragment of tissue examined. The number of *living* bacilli in the organs of these grouse is undoubtedly small; from which it is evident that they do not multiply in the organs. Grouse disease is therefore not an *infection* with these bacteria. Is it a toxaemia caused by the poison liberated from bacteria which have been absorbed from the intestine, and which have almost immediately perished in the tissue? We know that in order to produce serious mischief in animals by a single injection of dead bacteria a considerable quantity must be employed; and it is difficult to believe, when we remember the small numbers of colonies which grew on our cultures, that relatively to this quantity the numbers of bacteria absorbed could have been very large. On the other hand we have little information concerning the influence of the constant absorption of small numbers of bacteria, but this is believed by Adami and his school to be a potent source of disease. The fact that we have repeatedly found *B. coli* in the livers of "normal"

birds badly infected with strongyli prevents us from ascribing the death of the grouse directly to these bacilli, though they probably play some part.

Conclusions.

It seems to us quite certain that the strongylus when exceptionally numerous injures the mucous membrane of the caeca, and that this injury allows of the absorption of intestinal micro-organisms. It doubtless allows also of the absorption of other substances of an irritating or poisonous nature, and probably interferes with the normal selective absorption of nourishment. If we are right in thinking that the caecal contents become partly retained, and stick to the absorbing surfaces of the ridges of the mucous membrane, we have still more reason to believe that nutrition is greatly interfered with.

"Grouse disease," as we know it, appears to us not to be a specific bacterial infection. We conceive that all the birds which are more or less severely affected by strongyli suffer injury from this cause to an extent, which is more or less proportional to the severity of the infection. Some exceptionally strong birds may stand a larger infection better than weaker birds will stand a lesser; but, on the whole, the birds with the largest numbers of strongyli suffer most. Their nutrition gets impaired owing to interference with the normal absorption of digested food, and to the abnormal absorption of soluble poisons and intestinal bacteria. Such birds become the weakest; and when food becomes scarce, as it does at the beginning of spring, especially after bad winters or on overstocked moors, or when other harmful influences prevail, it is the weakest birds which suffer most. They die of privation acting on a constitution already weakened by the consequences of strongylosis, while their stronger neighbours manage to pick up a living somehow, and tide over the period of distress.

TABLE IX, *Summary of all observations.*

richostrongylus pergracilis—In Grouse Nos. 1—41 the strongyli were not counted, and only portions of the cæcal contents were examined.

In Grouse Nos. 43—68 (except 45) the strongyli were counted in both cæca separately (see p. 12).

In Grouse Nos. 69—81 (indicated * in Table) the strongyli were counted in one cæcum and the number found doubled.

umber	Date	Locality and History	Sex	Wt. in ozs.	Intestinal worms			Cultures from organs Liver
					Hymenolepis	*Davainea*	*Strongylus*	
1	5. v. 1908	Inverness. Caught unable to fly	♂	16	Numerous	–	Numerous	*B. coli* (12)
2	6. v. 08	,, ,, ,,	♂	16	,,	Numerous	,,	*B. coli* (several)
3	,,	,, ,, ,,	♂	—	,,	Few	,,	*B. coli* (12)
4	,,	,, ,, ,,	♂	20	,,	Numerous	,,	*B. coli* (few)
5	7. v. 08	Normal hand reared bird. Frimley	—	15	0	0	—	0
6	,,	Inverness. Caught unable to fly	♂	16	Numerous	Numerous	Numerous	*B. coli* (several)
11	9. v. 08	,, ,, ,,	♂	17	,,	,,	,,	*B. coli* ,,
12	,,	,, ,, ,,	♀	—	,,	,,	,,	*B. coli* (few)
13	,,	,, ,, ,,	♀	18	,,	0	,,	*B. coli* (1)
14	,,	,, ,, ,,	♂	16	,,	0	,,	*B. coli* (1) *st.c*
15	,,	,, ,, ,,	♂	23	,,	Numerous	,,	*B. coli* (several)
16	2. vi. 08	Normal. Frimley	♂	15	0	0	Few	–
17	10. vii. 08	,, ,, ...	♂	14	Few	1	Eggs only	*sb*
18	,,	,, ,, ...	♂	21	0	0	0	*sb*
19	24. vii. 08	,, ,, ...	♀	23	0	0	0	0
20	28. vii. 08	,, ,, ...	—	15	0	0	Few	–
21	5. viii. 08	,, ,, ...	♀	15	0	0	0	0
22	10. viii. 08	,, ,, ...	♀	—	0	2	Numerous	*B. coli* (1)
23	15. viii. 08	Inverness. Apparently healthy	♂	13	Numerous	Numerous	0	*B. coli* (4)
25	18. viii. 08	Normal. Frimley	♂	15	0	0	0	0
26	,,	,, ,, ...	♀	13	0	1	0	*S. lutea* (1)
28	—	Inverness	—	—	0	0	Numerous	*B. coli* (few)
29	27. viii. 08	,, Caught unable to fly ...	—	15	0	0	,,	*B. coli* (1)
30	26. viii. 08	,, Apparently healthy ...	♀	16	Few	Numerous	Few	0
31	27. viii. 08	,, ,, ,,	♀	20	Numerous	Few	Numerous	0
32	28. viii. 08	,, Healthy, caught on moor	♀	16	Moderate	Moderate	Moderate	*B. coli* (2)
33	,,	,, ,, ,, ,,	♂	15	Numerous	Numerous	Few	*B. coli* (few)
34	29. viii. 08	,, ,, ,, ,,	♂	18	Few	Moderate	,,	0
35	,,	Nairn. Caught on moor. Ill ...	♀	17	Numerous	Numerous	Numerous	*B. coli* (several)
36	3. ix. 08	Normal bird. Examined 4 days after death	♀	—	0	0	0	–
37	14. ix. 08	Normal bird. Found dead. Frimley	♀	11	0	0	0	–
38	23. x. 08	Normal bird. Died of pericarditis	♂	20	0	Numerous	Numerous	*st.c* (1)
39	24. x. 08	Lancashire. Caught unable to fly	♂	18	Few	,,	6230	–

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TABLE (*continued*).

B. enter. = Bacillus of the *B. enteritidis* group.
c = coccus.
m = mould.
sb = spore-bearing bacillus.
sx = streptothrix.
b = bacillus.
d = diphtheroid bacillus.
s = sarcina.
st.c = streptococcus.

The numbers or words in brackets indicate the number of colonies found.

Cultures from organs								
Lungs		Kidneys						
Right	Left	Right	Left	Spleen	Pancreas	Bile	Blood	Remarks
B. coli (1)	0	–	–	–	–	–	–	Examined 2 hours after death. Moderate reddening of cæcal villi.
d (3)	0	–	–	–	–	–	–	Cæcal mucous membrane much congested.
0	0	–	–	–	–	–	–	Cæcal mucous membrane little congested.
0	*stc* (2)	–	–	–	–	–	–	Cæcal mucous membrane not congested.
0	0	–	–	–	–	–	–	,, ,, ,, ,
0	0	–	–	–	–	–	–	—
m (1)	*b*	–	–	–	–	–	–	Cæcal mucous membrane much congested.
0	0	–	–	–	–	–	–	Cæcal mucous membrane slightly congested.
B. coli	–	–	–	–	–	–	–	Cæcal mucous membrane not congested.
0	0	–	–	–	–	–	–	,, ,, ,, ,,
B. coli	*B. coli*	–	–	–	–	–	–	Cæcal mucous membrane extremely congested.
0	–	–	–	–	–	–	–	Ditto, not congested. ? Gut wounded while making cultures.
sx *sb*	*sx* *sb*	*sb*	*sb*	*sb* *m*	–	0	0	Cæcal mucous membrane not congested.
sb	*sb*	*sx*	0	0	–	*sb*	0	,, ,, ,, ,,
0	0	0	0	*sb* (1)	–	0	0	—
0	0	0	0	–	–	–	–	Gut wounded when making cultures.
m (1) *sb* (1)	0	0	0	0	–	0	–	—
sb (1)	*m* (1) *c* (1)	*B. coli* (2)	0	0	–	0	–	—
0	*m* (1)	*sb* (1)	0	*B. coli* (1)	–	0	–	Numerous portions of *Davainea* in cæca.
m *sx*	*m* *sb*	0	*c* (1)	0	–	0	–	—
m (1) *d* (1)	*m* (1) *sx* (1)	0	0	0	–	0	–	—
m (2)	*sb*	0	0	0	–	–	*c* (1)	Shot in wing. Brought in alive.
m *sx*	*sx* (1)	0	0	0	–	–	–	—
m (1)	*m* (1)	*sb* (1)	0	0	–	–	–	—
m (1) *sb* (1)	*m* (1) *sb*	0	0	*B. coli* (1)	–	–	–	—
m (3)	*m* (1)	*sb* (1)	0	0	–	–	–	—
0	*sb* (1)	0	0	*s* (1)	–	–	–	—
0	0	*sb* (1)	0	0	–	–	–	—
0	*b* (1)	0	0	0	–	–	0	Cæcal mucous membrane somewhat reddened.
–	–	–	–	–	–	–	–	—
–	–	–	–	–	–	–	–	—
0	0	0	0	–	–	–		
–	–	–	–	–	–	–	–	Arrived dead.

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TABLE (*continued*).

Number	Date	Locality and History	Sex	Wt. in ozs.	Intestinal worms			Cultures from organs Liver
					Hymenolepis	*Davainea*	*Strongylus*	
40	28. x. 1908	Normal. Died of pneumonia. Frimley	♂	20	0	0	Few	0
41	,,	Lancashire. Caught unable to fly	♂	20	0	Moderate	Numerous	–
43	8. xii. 08	Cumberland. Normal ...	♂	17	0	1	45	0
44	,,	,, ,, ...	♀	19	0	0	871	*B. coli* (4)
45	,,	Nairn. Picked up dead ...	♂	16	0	Moderate	Numerous	–
46	9. xii. 08	Cumberland. Normal ...	♂	16	0	0	259	*B. coli* (1)
47	,,	,, ,, ...	—	18	0	0	344	*B. enter.* (several)
48	,,	,, ,, ...	♂	—	0	0	32	*s* (2)
49	,,	,, ,, ...	♀	13	0	Several	330	*c* (1)
50	,,	,, ,, ...	♀	19	0	0	290	*B. coli* (3)
51	,,	,, ,, ...	♀	13	0	0	540	*B. coli* (many)
52	21. xii. 08	Lancashire. Normal. Hand reared	♂	15	0	0	0	0
53	—	Montgomeryshire. Picked up dead	♂	16	0	0	2506	–
54	3. ii. 09	Cumberland. Normal ...	♀	16	0	6	1645	*B. coli* (several)
55	,,	,, ,, ...	♂	17	0	7	415	0
56	5. ii. 09	,, ,, ...	♂	16	0	5	1868	*B. coli* (many)
57	,,	,, ,, ...	♀	17	0	11	599	*B. coli* (several)
58	,,	,, ,, ...	♀	17	0	0	113	0
59	6. ii. 09	,, ,, ...	♀	12	0	0	235	*B. coli* (several)
60	17. iii. 09	Yorkshire. Normal. Caught on moor	♀	21	Few	Numerous	5995	*B. coli* (several)
61	,,	,, ,, ,, ,,	—	21	Numerous	Numerous	9562	*B. coli* (several)
62	19. iii. 09	,, ,, ,, ,,	♀	21	0	0	730	*B. coli* (8)
63	20. iii. 09	Selkirk. Normal. Caught on moor	♀	18	Numerous	0	533	*c* (1)
64	,,	,, ,, ,, ,,	♀	20	,,	Numerous	571	0
65	,,	Cumberland. Shot ...	♀	18	,,	2	183	–
66	,,	Selkirk. Normal. Caught on moor	♀	19	,,	1	945	0
67	,,	,, ,, ,, ,,	♀	18	,,	Moderate	833	0
68	22. iv. 09	Caithness. ,, ,, ,,	♀	16	0	0	877	–
69	27. iv. 09	Inverness. ,, ,, ,,	♂	23	Numerous	Numerous	730*	–
70	,,	,, ,, ,, ,,	—	23	,,	,,	2524*	–
71	10. v. 09	,, Caught, unable to fly	♀	16	,,	0	7058*	–
72	,,	,, Picked up dead. ...	♂	17	,,	Numerous	10266*	–
73	7. v. 09	Nairn. ,, ,, ...	♀	17	,,	Fragments	7484*	–
74	21. v. 09	Yorkshire. ,, ,, ...	♀	17	0	Moderate	3114*	–
74 (*a*)	,,	,, ,, ,, ...	—	—	Numerous	0	3406*	–
75	19. v. 09	,, ,, ,, ...	♂	—	,,	Numerous	4352*	–
76	,,	Lancashire. ,, ,, ...	—	—	0	,,	18332*	–
77	,,	Selkirk. ,, ,, ...	♂	21	Numerous	0	8800*	–
78	24. v. 09	,, ,, ,, ...	♀	16	,,	Few	3340*	–
79	3. vi. 09	Inverness. ,, ,, ...	♀	17	Few	0	2556*	–
80	,,	,, ,, ,, ...	♂	19	0	0	3840*	–
81	17. xii. 09	Normal. Frimley	♀	15	0	0	0	0

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TABLE (*continued*).

Cultures from organs								
Lungs		Kidneys						
Right	Left	Right	Left	Spleen	Pancreas	Bile	Blood	Remarks
sx (1) *sb* (1)	*m* (1)	–	–	0	–	0	0	—
–	–	–	–	–	–	–	–	Arrived dead.
sb (1)	*st.c* (1) *m* (1) *sx* (1)	0	0	–	–	–	–	—
m (1) *c* (1)	*m* (1)	0	0	0	0	–	0	—
–	–	–	–	–	–	–	–	—
sx (1) *m* (1)	*sx*	0	0	0	–	–	0	—
m (1) *c* (1)	*m* (1) *c* (1)	0	*sb* (1)	*B. enter.*	–	–	–	Patches of dry fæcal matter adhering to cæcal wall.
0	*sx*	0	*sb*	–	0	–	–	—
sx *m*	*m*	*c* (1)	0	0	0	–	–	—
B. coli (1) *m* (1)	*m* *sx*	0	0	*B. coli* (6)	0	–	–	—
sx *m*	*sx* *m* *B. enter.*	*B. enter.*	0 *B. enter.*	*B. enter.*	*B. enter.*	–	–	Bird in very poor condition.
m *c*	*m* *c*	0	0	0	0	–	–	32 specimens of *Trichosoma longicolle* in duodenum. 32 specimens of *Heterakis papillosa* in cæca.
–	–	–	–	–	–	–	–	2 specimens of *Trichosoma longicolle* in duodenum.
m	*m*	0	0	*B. coli* (3)	...	...	...	—
m	*sx* *m*	0	0	–	*s* (1)	–	–	—
B. coli *m* (1), *c* (1)	*c*	–	0	–	–	–	–	—
m	*m*	0	–	0	0	–	–	—
m	*sx* *m*	–	*s* (1)	0	–	–	–	—
m *sx*	*sx*	0	0	0	–	–	–	1 specimen of *Heterakis papillosa* in each cæcum.
0	*m* (1)	0	–	–	–	–	0	,, ,, ,, ,,
0	0	0	0	0	*s* (1)	–	0	—
sx *m*	*sx* *m*	0	0	–	0	–	0	—
sx	*sx*	*c* (1)	0	0	0	–	0	—
0	0	0	0	0	0	–	0	—
–	–	–	–	–	–	–	–	—
m (1)	*m* (1)	0	0	–	0	–	0	—
m (1)	0	0	0	0	0	–	0	—
–	–	–	–	–	–	–	–	Arrived dead.
–	–	–	–	–	–	–	–	,,
–	–	–	–	–	–	–	–	,,
–	–	–	–	–	–	–	–	
–	–	–	–	–	–	–	–	—
–	–			–	–	–	–	—
–	–	–	–	–	–	–	–	—
–	–	–	–	–	–	–	–	—
–	–	–	–	–	–	–	–	—
–	–	–	–	–	–	–	–	—
–	–	–	–	–	–	–	–	—
	–	–	–	–	–	–	–	—
–	–	–	–	–	–	–	–	—
–	–	–	–	–	–	–	–	—
m	*m*	0	0	0	–	0	0	—

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EXPLANATION OF PLATES I—V.

Fig. 1. ($\frac{1}{4}$ nat. size) showing the glass frame in which the crushing of the tissues was done. On the ledge are three pairs of ground glass plates. One of the plates of the pair on the left is being held up in the manner in which this was done preparatory to placing a piece of tissue between the plates.

Fig. 2. ($\frac{1}{3}$ nat. size) showing the alimentary canal of a normal grouse (No. 81) from the gizzard to the anus. Gizzard (*a*), duodenum, enclosing the pancreas (*b*), intestine (*c*), caeca (*d*, *d*) and rectum (*e*).

Fig. 3. ($\frac{1}{2}$ nat. size) showing small test tubes arranged in pairs containing the strongyli collected from the two caeca of eleven grouse. The serial number of the grouse is written on the top, and the number of strongyli present in each tube in smaller figures below.

Fig. 4. ($\frac{1}{2}$ nat. size) showing small test tubes containing the strongyli collected from both caeca of 18 grouse. No strongyli were found in the caeca of grouse No. 52, and in this case the test tube contains 32 specimens of *Heterakis papillosa*. The serial number of the grouse is printed above each tube.

Fig. 5. (×9) showing a transverse section of the caecum of a normal grouse (No. 81). The ridges (*a*) and villi (*b*) are well shown, and three collections of lymphoid tissue (*c*) are also seen.

Fig. 6. (×25) showing a portion of the same section more highly magnified.

Fig. 7. (×25) showing a portion of a section of the caecum of an apparently healthy wild grouse (No. 69).

Fig. 8. (×9) showing a transverse section of the caecum of a diseased grouse (No. 2).

Fig. 9. (×9) showing a transverse section of the caecum of a diseased grouse (No. 15) very badly infected with strongyli.

Fig. 10. (×25) showing a portion of Fig. 9 more highly magnified. Large numbers of worms are seen in transverse section as black dots.

Fig. 11. (×5) showing the internal surface of the caecum of a normal grouse (No. 81) after gentle washing. Several ridges are seen, some of which die away near the centre of the specimen. The whole surface is covered with small villi.

Fig. 12. (×5) showing the internal surface of the caecum of an apparently healthy wild grouse (No. 69) after gentle washing. The ridges are greatly developed, and the villi larger and more prominent than in the preceding figure. A few worms can be seen.

Fig. 13. (×5) showing the internal surface of the caecum of a diseased grouse (No. 73) after gentle washing. The ridges are very broad, and the villi in some places hypertrophied (*a*). In one situation the villi are so matted together that they are almost indistinguishable (*b*). At this spot a mass of dry material adhered to the ridge.

Fig. 14. (×5) showing the internal surface of the caecum of a diseased grouse (No. 12) after gentle washing. The ridges are very prominent, but the villi are matted together to such a degree with cementing material that they are almost indistinguishable. Some of the ridges are united with the same material (*a*, *b*, *c*).

Fig. 15. (×100) showing two specimens of *T. pergracilis* (*a*, *b*) in section in the epithelium covering a villus.

Fig. 16. (×100) showing a specimen of *T. pergracilis* (*a*) in section surrounded by a ring of fibrous tissue (*b*).

Fig. 17. (×5) showing large numbers of *T. pergracilis* on the internal surface of the caecum of a diseased grouse (No. 13).

Fig. 18. (×100) showing a specimen of *T. pergracilis* (in section) between two villi. The epithelium has been lost and fibrous tissue (*a*) has been formed within one of the villi in the neighbourhood of the worm.

Fig. 19. ($\frac{2}{3}$ nat. size) showing two tubes containing the specimens of *Davainea urogalli* (on the left) and of *Hymenolepis microps* (on the right) obtained from grouse No. 11.

Fig. 20. (nat. size) showing a tangled mass of *Davainea urogalli* (partially opened out) from the intestine of a grouse.

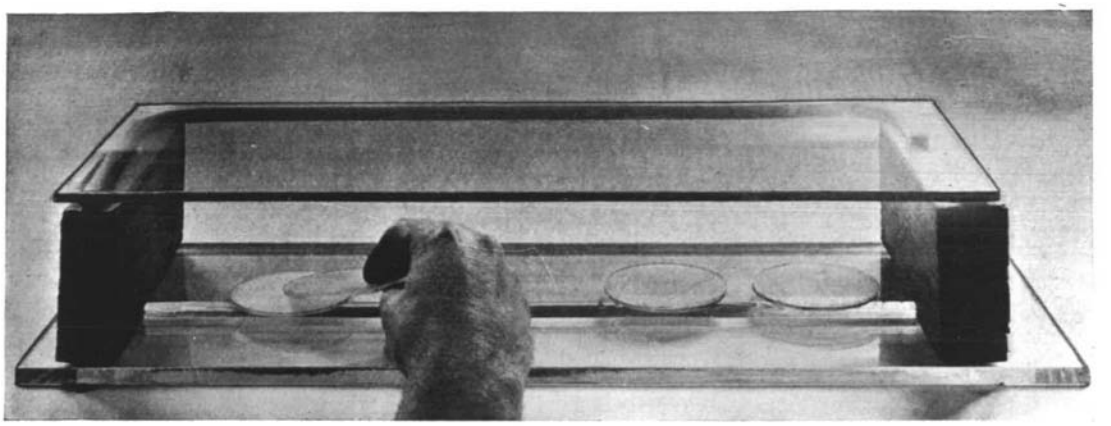

Fig. 1.

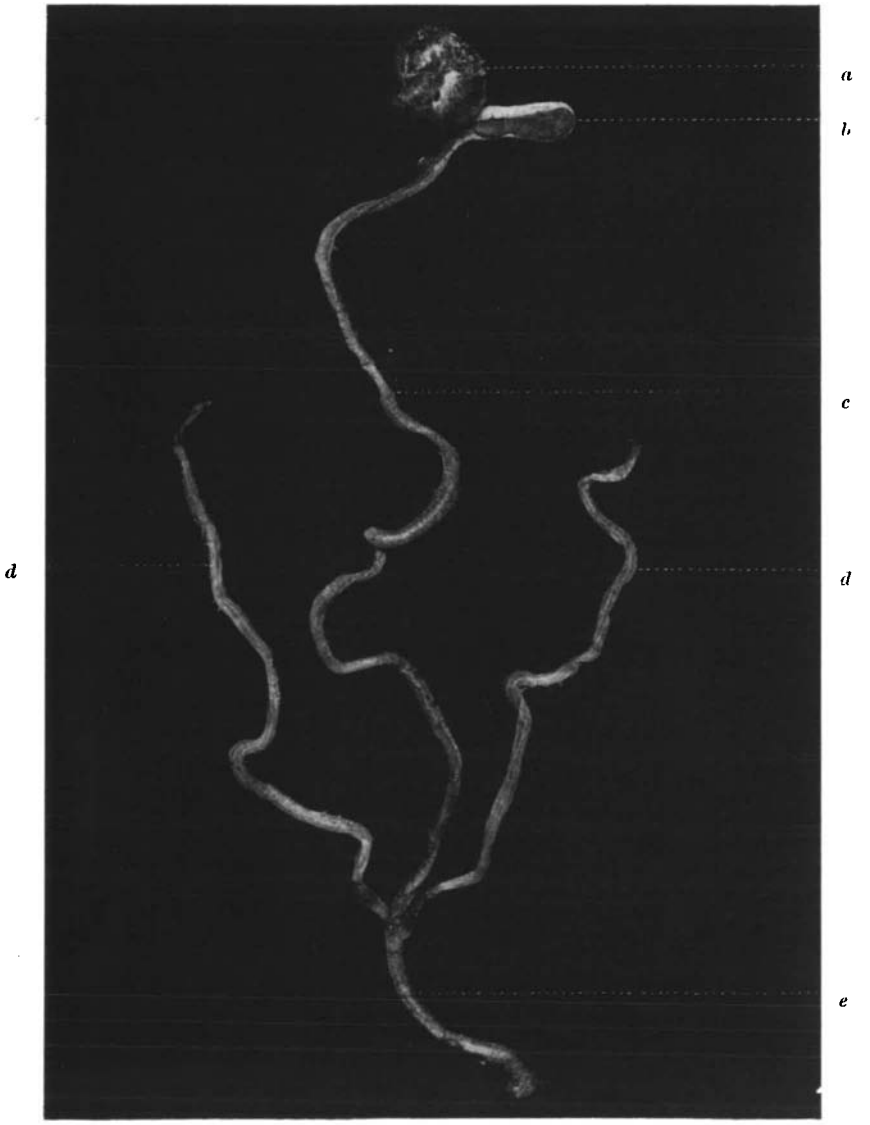

Fig. 2.

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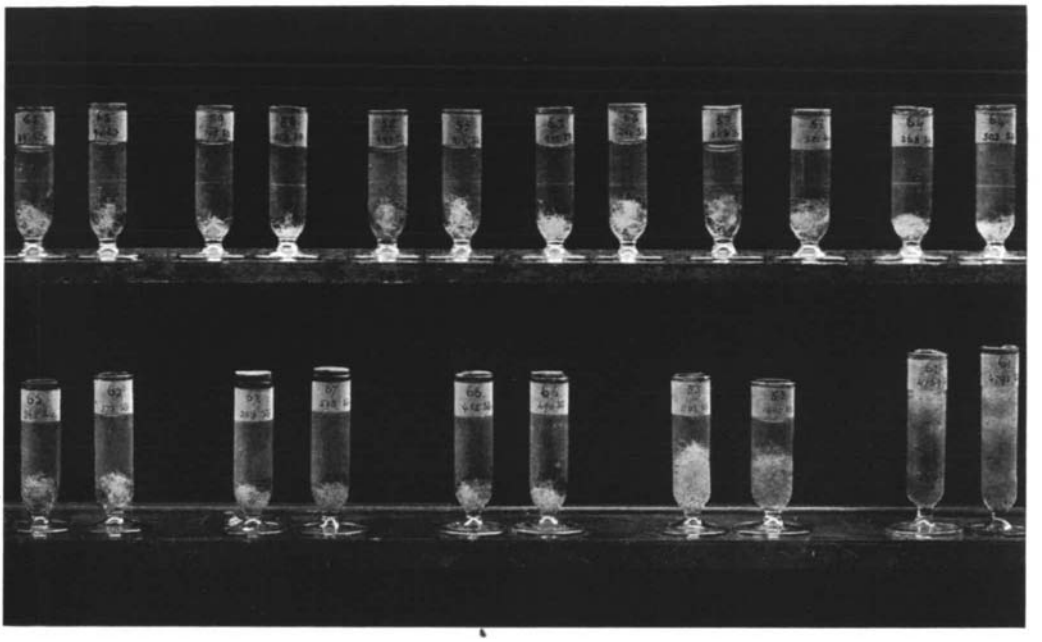
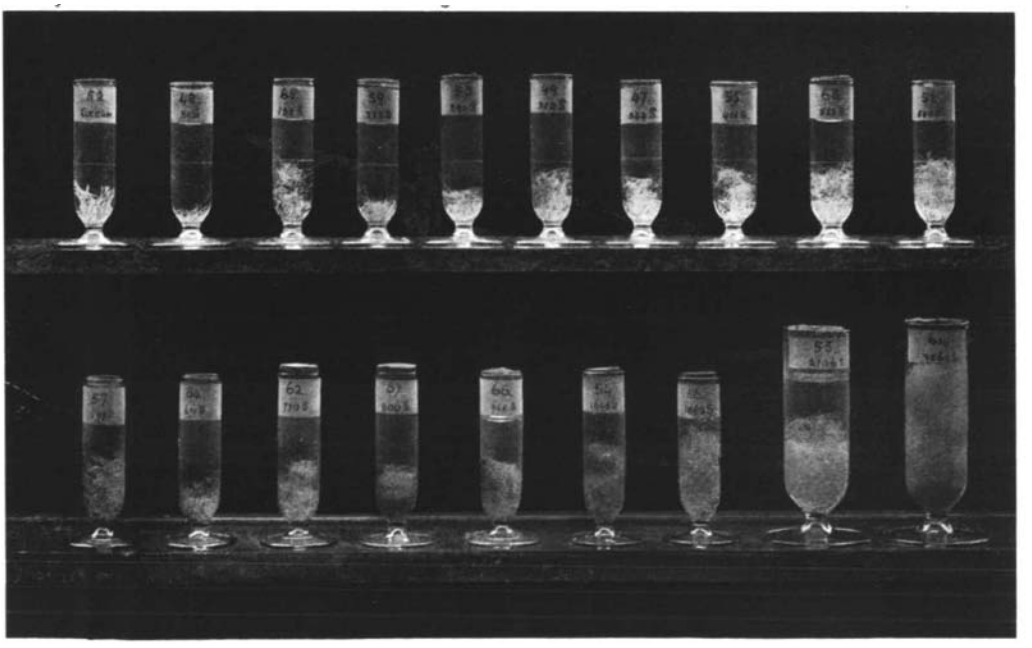
65 (89—94) 59 (107—108) 55 (201—214) 63 (252—281) 57 (268—331) 64 (268—303)



62 (365—375) 67 (258—548) 66 (455—490) 53 (1103—1403) 61 (4769—4793)

Fig. 3.

52 48 65 59 50 49 47 55 63 51



57 64 62 67 66 54 56 53 61

Fig. 4.

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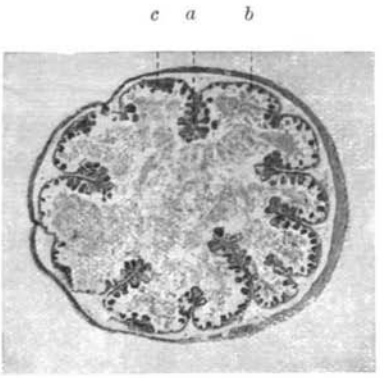

Fig. 5.

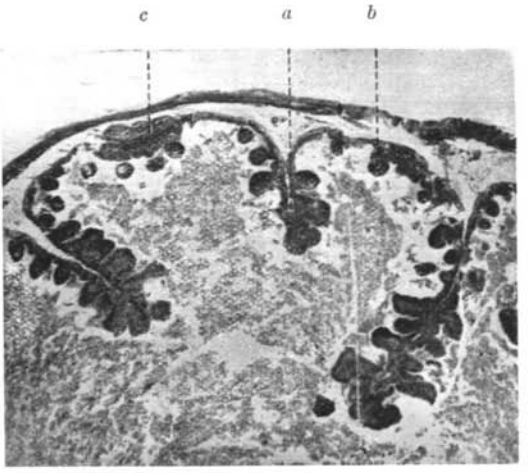

Fig. 6.

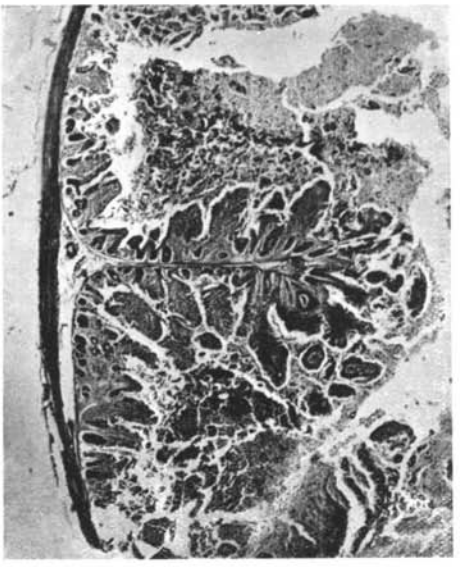

Fig 7.

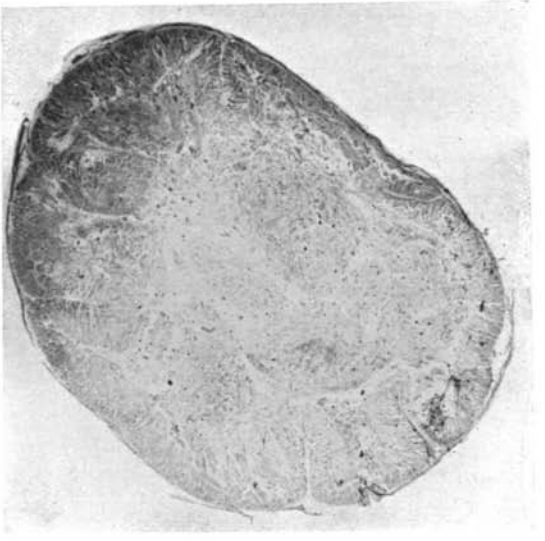

Fig. 8.

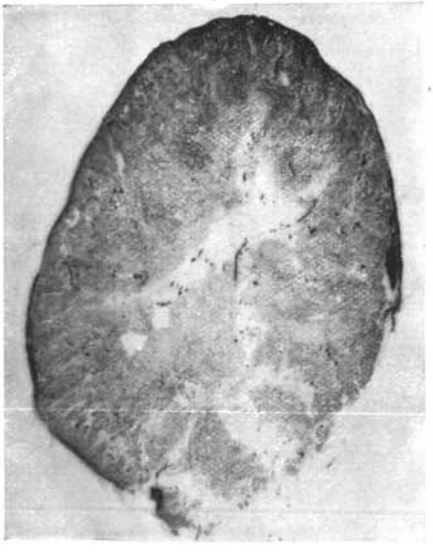

Fig. 9.

Fig. 10.

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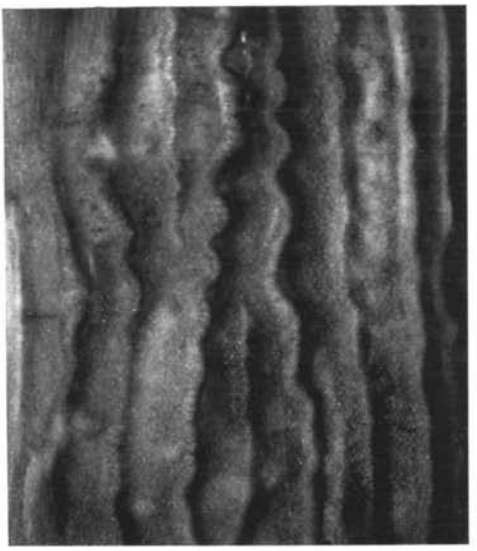

Fig. 11.

Fig. 12.

Fig. 13.

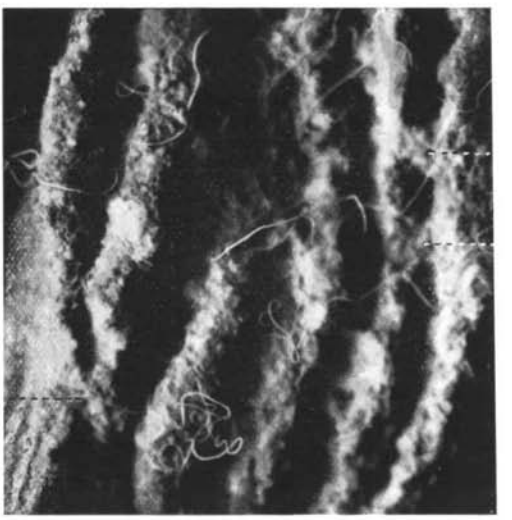

Fig. 14.

Fig. 15.

Fig. 16.

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Fig. 17.

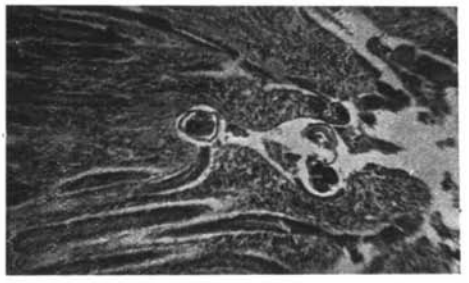

Fig. 18.

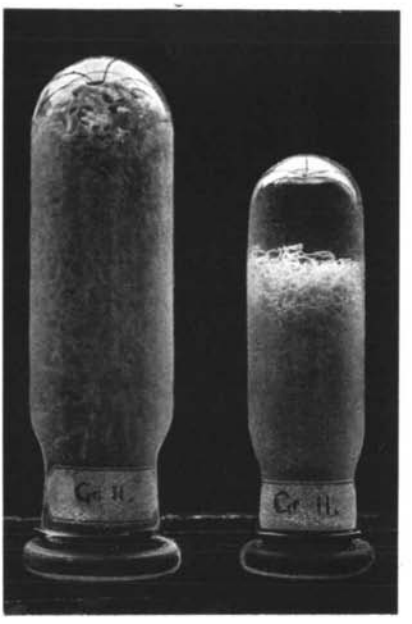

Fig. 19.

Fig. 20.

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