Causes of mortality and culling in adult pheasants

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The causes of the deaths or culling of 155 adult pheasants in breeding pens on one site between 1995 and 1997 were investigated. Approximately half the deaths were the result of problems associated with the reproductive tract or trauma, including injuries acquired during fighting or matting. Sinusitis was the commonest infectious cause of mortality or culling, despite medication of the flocks for mycoplasmosis. Marble spleen disease and pheasant coronavirus-associated nephritis, two viral conditions capable of causing high mortality, were diagnosed in a few birds in 1996 and 1997. Histomoniasis (blackhead) contributed to the mortality in 1996. A lymphomatous condition of uncertain aetiology was detected in a small number of birds.

APPROXIMATELY 12 to 15 million pheasants (Phasianus colchicus) are shot for sport in the UK each year (National Game Dealers Association, unpublished data). This number of birds cannot be provided by the population of wild pheasants, of which there are approximately three million breeding birds each spring (Gibbons and others 1993), so the numbers are supplemented by artificially reared pheasants. It is therefore a common practice for pheasant-rearing sites to catch pheasants from the wild each year in February and March and transfer the birds to static or moveable breeding pens, some of which will hold over 100 hen pheasants plus smaller numbers of cock pheasants (Anon 1993). Most of these birds are approximately eight to 10 months of age when caught. The fertile eggs produced by these breeding flocks are incubated artificially, and the chicks which hatch are reared and released when six to eight weeks old (Anon 1983).

While they are in the breeding pens, the adult pheasants are susceptible to a variety of infectious and non-infectious causes of disease, and a mortality level of 3 per cent is considered acceptable for the five months they spend in the breeding pens (Wise 1993). Investigations are usually carried out only if there is an unacceptably high level of mortality and the underlying causes of the mortality are not immediately obvious. Nevertheless, it is important for several reasons that these investigations are made. Closer examination of ‘normal’ flocks could help to establish approximate guides to mortality rates during the different periods spent in the breeding pens, which, if exceeded, would indicate the need for further investigations. This information could also indicate ways in which changes in husbandry or prophylactic medication might reduce mortality rates, and add to the understanding of the epidemiology of certain infectious diseases of pheasants and other domesticated livestock.

The causes of death or culling of adult pheasants on one gamebird rearing site were therefore monitored from 1995 to 1997.

MATERIALS AND METHODS

Site and birds

The adult pheasants were monitored in 1995, 1996 and 1997 on a commercial rearing enterprise in south-west Scotland which had pens for breeding birds, incubation facilities, rearing pens and release pens. Beginning around the third week of February in each year, semi-wild female pheasants were caught from the estate and several other estates within a 75 mile radius over a period of three to four weeks. Most of these birds had themselves been reared on the site and then released on to the estates six to eight months earlier. Smaller numbers of male pheasants were also caught on the estates, but the majority of the male pheasants were purchased from a different site in northern England. Following standard practice (Wise 1993), plastic ‘spectacles’ were fitted to all the birds to reduce egg eating and cannibalism, ribbon brails were applied to one wing to reduce mobility, and the spurs of the males were blunted to reduce damage to the females.

The pheasants were accommodated in a series of adjoining grass pens with netted sides and roof. Each pen measured 21.9 m by 14.6 m and held 60 to 62 female pheasants and eight to nine males, giving approximately 4–6 m² per bird. In 1995, 1200 female pheasants and 160 male pheasants were placed; in 1996, 1240 females and 160 males; and in 1997, 1580 female and 220 male pheasants. In 1996 the pens were kept on the site first used in 1995, but in 1997 a different site was used. Each pen contained one weatherproof food hopper, one automatic drinker connected to a common outdoor header tank, and half barrels were provided as nest sites.

All the birds received the in-feed anthelmintic fluben-dazole (Flubenvet; Janssen Animal Health) for five to seven days, approximately three to five weeks after the breeding pens began to be filled, and the treatment was repeated as necessary during the following months of egg production. To reduce the number of cases of mycoplasmosis, tiamulin (12.5 per cent Tiamulin; Leo Laboratories) was administered periodically in the drinking water, and in 1997, live Newcastle disease vaccine (Poulvac Hitchner B1; Fort Dodge Animal Health) was administered by eyedrop to each bird. A proprietary breeder pellet containing 18 per cent crude protein was provided ad libitum from when the birds were caught to the end of egg collection, some 18 to 19 weeks later.

Arrangements were made with the gamekeeper in charge of the site for all the birds which died or were culled in the breeding pens to be submitted for postmortem examination.

Production periods

The time spent in the breeding pens was divided into four unequal periods as follows: period 1, from the start of catching up to the onset of egg production, approximately six weeks; period 2, the first four weeks of egg production; period 3, the second four weeks of egg production; and period 4, the remaining period to depopulation, approximately four to five weeks.

Postmortem examination

A standard postmortem examination was carried out on each carcase, including a gross examination of all major organs and the preparation and microscopical examination of wet smears from the crop, small intestine and caecum. Bacteriological, virological and histopathological investigations were carried out, if required, to establish the cause of death or culling.
The carcasses of 155 birds (139 females and 16 males) were examined. In 1996 and 1997 the overall mortality was a little over 4 per cent for the period of 18 to 19 weeks spent in the breeding pens (Table 1). This figure is similar to that suggested by Wise (1993), who considered breeding pen mortality to be acceptable if below 5 per cent. The mortality recorded in 1995 was lower but not representative, because not all the carcasses were submitted owing to logistical problems that were resolved in 1996 and 1997.

Taking the 1996 and 1997 figures as representative, it was calculated that one to two birds per thousand died or were culled per week in period 1, two to three birds per thousand per week in period 2, and three to four birds per thousand per week in periods 3 and 4, resulting in a final mortality rate of a little over 4 per cent for the 18 to 19 weeks. This breakdown provides useful figures against which gamekeepers and their veterinary advisers can assess pheasant mortality.

Infectious diseases (excluding lymphomatous tumours which may have a viral aetiology) accounted for the deaths of 49 birds, that is, 31.6 per cent of the carcasses examined (Tables 2, 3). Reproductive tract disorders were responsible for the deaths of 43 birds (27.7 per cent of the carcasses), and 35 birds died of trauma (22.6 per cent of the carcasses). Eight birds (5.2 per cent of the carcasses) died or were culled as a result of lymphomatous tumours, and the remaining 20 birds (12.9 per cent of carcasses) died or were culled for other miscellaneous reasons.

**Sinusitis**

Thirteen birds died or were culled with sinusitis. The affected birds showed a combination of upper respiratory tract lesions, such as conjunctivitis, periorbital loss of feathers, and distension of the infraorbital sinuses with muccoid or purulent material. In some birds similar material also filled the nasal passages and extended through the choana into the mouth. In addition to the upper respiratory tract lesions, one bird had multiple pale granulomata in the lungs; another had similar granulomata in the lungs, liver and spleen; a third had pericarditis and periphlebitis; and a fourth had marked splenomegaly and hepatoatony, with histopathological changes of septicaemia. Bacterial cultures from the sinuses yielded mixed growths of bacteria including *Escherichia coli*, *Enterococcus faecalis*, *Pseudomonas aeruginosa*, and *Pasteurella haemolytica*. *E coli* was also isolated from the viscera of the bird with pericarditis and periphlebitis and from the lungs of the bird with a granulomatous pneumonia, and *P haemolytica* was recovered from the bird with splenomegaly and hepatoatony. However, no significant bacteria were isolated from the viscera of the bird with granuloma in its lungs, liver and spleen. No cultures for mycoplasma were made.

**Nephritis**

Ten birds, one in 1996 and nine in 1997, had enlarged and pale kidneys, with one or both ureters impacted with urate casts, and urates on the surface of their livers and hearts. All the affected birds were female, and eight of them died between the start of the second month of egg production and depopulation (periods 3 and 4). Histologically, the kidney tubules were dilated and the collecting ducts were distended with casts made up of necrotic cells and granulocytes. These changes were often severe, with areas of tubular necrosis, mineralisation, and an interstitial infiltration of mononuclear inflammatory cells.

This condition, usually referred to as 'pheasant coronavirus-associated nephritis', 'pheasant coronavirus nephritis' or 'pheasant urolithiasis' was first recorded in Hampshire in 1983 (Lister and others 1985) and has since spread to many other areas of the UK. Ten years after the disease was diagnosed in southern England, it was diagnosed at two sites in southwest Scotland in 1993, one of which was the site described in this study. A virus designated '750/83' has been isolated from the tissues of pheasants with such lesions and has the appearance of a coronavirus when examined by an electron microscope (Lister and others 1985, Gough and others 1996). This virus is different from strains of coronavirus recovered from poultry with infectious bronchitis (including strains M41, 793/B, D274 and D1466) and has been termed 'pheasant coronavirus' (Lister and others 1985, Gough and others 1996). Although it has been suggested that the initial kidney damage occurs earlier in life, with kidney failure precipitated later by additional factors such as chilling, water deprivation, a change of accom-
modation, and the requirement to mobilise calcium for egg shell production (Lister 1989). No trigger factor could be identified in the present study, in which most of the losses occurred in the last few weeks of egg production.

A mortality rate exceeding 14 per cent was reported by Gough and others (1996), and there are anecdotal reports of much higher mortality in some flocks. However, in the present study, the condition had become endemic, and there were few deaths.

**E coli septicaemia**

In 1996 a fibrinous pericarditis and peripneumonia was observed in two birds from which heavy growths of *E coli* were recovered. In one bird the condition was secondary to an upper respiratory tract infection, and in the other the precipitating factor was considered to be an egg yolk peritonitis. In 1997, colisepticaemia was diagnosed in six birds in the absence of respiratory tract or reproductive tract disorders. All the affected birds had enlarged livers, with small foci of liver necrosis in some birds. Five of the six birds also had enlarged spleens, which were dark in colour in some birds, and with fine necrotic foci in one bird. Bacterial cultures from all the birds yielded *E coli* in septicaemic distribution. Histopathological examination of visceral organs confirmed the presence of a septicaemia, but did not indicate any primary condition which could have predisposed the birds to the colisepticaemia. The agar gel immunodiffusion test (AGIT) for marble spleen disease was negative in all the affected birds. These losses from colisepticaemia occurred when other birds were dying from pheasant coronavirus-associated nephritis, and may have been secondary to exposure to this virus; this could not be confirmed because no virological studies were carried out.

**Marble spleen disease**

Marble spleen disease was diagnosed in six birds in 1996, and one bird in 1997. The deaths in 1996 occurred over a period of five weeks, starting about one week before the onset of egg production, a period during which six out of 10 birds dying had the condition. In 1997, the only bird to be affected died seven weeks after the onset of egg production. All the birds were female, had been eating, were in good body condition, and had very congested lungs postmortem. In most of the birds the spleen was enlarged and pale, but in two birds the spleen appeared normal. The diagnosis was confirmed by the demonstration of large, faintly basophilic, intranuclear inclu- sions bodies in the reticulendothelial cells of the spleen, or by demonstration of viral antigen in the spleen by the AGIT.

Marble spleen disease was first diagnosed in the UK in 1972, in three-month-old poults found dead in good body condition (Bygrave and Patissom 1973). Since then, the virus appears to have become widespread in pheasants (Wise 1993), with mortality most often occurring in adult birds in breeding pens. The condition is caused by a type II avian adenovirus (Fitzgerald and Reed 1989), related to the virus which causes haemorrhagic enteritis of turkeys. Typically, birds are found dead with no premonitory signs, although respiratory signs may occur immediately before death (Fitzgerald and Reed 1989). A combination of severe lung congestion and an enlarged, pale or marbled spleen is considered to be typical of the disease (Fitzgerald and Reed 1989), but in the present study, although two of the affected birds had very congested lungs, the spleens were of a normal appearance. The possibility of marble spleen disease should therefore be considered if pheasants are found dead in good body condition and with congested lungs, but with grossly normal spleens.

The epidemiology of the disease is unclear. Mortality can exceed 50 per cent (Wise 1993), but in the present study the losses due to the disease were very low, and all the affected birds were found in one pen, in birds from one source, and there was no mortality in adjacent pens. It has been suggested that mortality from the disease is precipitated by stress, and in the cases observed in 1996, the precipitating factor may have been the onset of egg production.

Vaccines developed from the related haemorrhagic enteritis virus of turkeys have been shown to protect pheasants against the disease (Fitzgerald and Reed 1989), but in the absence of such a vaccine in the UK, its control must be based on reducing stress factors to a minimum.

**Helmints**

Of the 155 pheasants examined, *Syngamus trachea* was found in 13 birds, *Heterakis species* (most likely *Heterakis gallinarum*) in 23 birds, *Eucoleus* (*Capillaria*) species in nine birds, and *Trichostrongylus* species in two birds. Most of the *Eucoleus* species worms or eggs were found in the upper digestive tract, suggesting that the species was *Eucoleus annulatus*. No nematodes of the genus *Ascaridia*, and no cestodes or trematodes were found. Eighteen of the 155 birds were examined before they had been wormed; among these, *S trachea* was found in two, *Heterakis* species in seven, and *Eucoleus* species in eight. In contrast, in the months after the birds had been wormed, *Eucoleus* species were not found, being found in only one of the 137 birds examined: *Heterakis* species was found in 12 per cent of the birds examined four to eight weeks after worming; and *Syngamus* species was found in 8 per cent of the birds, positive birds being examined three to 10 weeks after worming. In most of the birds the parasites were not considered to be significant, but in three birds the burdens of *Eucoleus* species were associated with poor body condition requiring them to be culled, and in three other birds the burdens of *Syngamus* species were the cause of death or culling. *Heterakis* species were not considered to be of primary significance in any of the birds, but may have been important in the transmission of histomoniasis (see below).

Most gamekeepers are familiar with the gapeworm *S trachea*, because the clinical signs of coughing, sneezing, head shaking and gapeing in affected birds are obvious. However, the results of this study suggest that pheasants caught up after spending several months in the woods and fields are more likely to have acquired worms of the genera *Eucoleus* and *Heterakis* than *Syngamus*. Caught-up birds should therefore be wormed as soon as possible, even in the absence of signs of gapeworm burdens.

**Burdens of *Heterakis* and *Syngamus* species were rapidly reacquired from the pasture of the breeding pens, sometimes within three to four weeks of worming, and regular administration of an anthelmintic may be necessary when birds are in their breeding pens. Both *Syngamus* and *Heterakis* species have direct life cycles, although invertebrates such as earthworms, snails, slugs and flies can act as transport hosts for *Syngamus* species, and earthworms as transport hosts for *Heterakis* species (Soulby 1982). However, *Eucoleus* species were rarely found in birds in the breeding pens after they had been wormed. There is some debate as to the identity and life cycle of species of *Eucoleus* found in the upper digestive tract of birds, and it is unclear whether the low recovery of *Eucoleus* species after the initial worming reflects an indirect life cycle, a longer period of development in the bird, or greater difficulty in detecting this small worm and its eggs. *Trichostrongylus* eggs, presumably of the type *Trichostrongylus tenius*, were found in small numbers in the caeca of two pheasants. *T tenius* has been linked with poor breeding success and population cycles in red grouse (*Lagopus lagopus scoticus*) (Hudson 1986, Potts and others 1984), and in the past with disease in grey partridge (*Perdix perdix*) (Potts 1986). However, these parasites do not appear to cause clinical disease in pheasants, and were not considered to be significant in the present study.

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Histomoniasis

Histomoniasis (blackhead) was diagnosed in six female pheasants over a period of four weeks in 1996. Most of the affected birds were in good body condition, but had multiple coalescing yellow or fawn circular areas up to 15 mm diameter on the surface and cut surfaces of the liver. The centres of the lesions were often slightly darker and more depressed than the surrounding areas of liver necrosis. Only one of the six birds had gross areas of the caeca, with one caecum having a thickened wall, mucosal petechiae and white cores of inflammatory debris. Wet smears were prepared from the affected livers and caeca and examined microscopically but no motile protozoa were observed, probably because of the interval between the death of the birds and the examination of the carcasses. No significant bacteria were isolated or demonstrated on aerobic or anaerobic cultures, or on Ziehl-Neelsen or Gram-stained smears from affected areas. Histopathological examination of affected livers showed coalescing areas of hepatocyte necrosis, surrounded by mononuclear cells and often giant cells, and in some birds there were periodic acid-Schiff (PAS)-positive protozoa, presumed to be *Histomonas meleagrisidis*, at the periphery of the lesions. Similar organisms were also found in PAS-stained sections of the intestinal tracts of the birds.

Histomoniasis is a parasitic disease of the caeca and liver of many gallinaceous birds including the turkey, domestic fowl, peafowl, guineafowl, chukar partridge and pheasant (McDougald 1991). The causal organism, *H. meleagrisidis*, is initially found in the caeca, but can reach the liver by the hepatic portal system (Soulsby 1982). In young turkeys the disease is characterised by the production of sulphur-yellow diarrhoea, anorexia and death, with postmortem lesions including distension of the caeca with caseous or haemorrhagic cores, ulceration and sometimes perforation of the caeca, and circular depressed areas of liver necrosis (McDougald 1991). There is marked variation in the susceptibility of different species of birds to histomoniasis. Lund and Chute (1972a, b) found that turkeys, chukar partridges and peafowl were most susceptible, followed by guineafowl and domestic fowl, with the pheasant being considerably less susceptible. Nevertheless, because of the potential risk from this and other protozoal conditions, it is standard practice to incorporate the antiprotozoal agent dimetridazole into the feed of growing pheasants (Wise 1993).

In view of the relatively low susceptibility of pheasants to histomoniasis, there must have been a heavy challenge to cause the liver lesions observed in the birds observed in turkeys. The good body condition and absence of caecal lesions in most of the birds suggest that histomoniasis can be an acute disease in pheasants.

Free histomonads cannot survive outside the host for more than a few minutes (McDougald 1991). However, histomonads can infect the eggs of the parasitic nematode *H. gallinarum* which is also found in the caeca and whose eggs are subsequently passed in the faeces. The histomonads survive in the *Heterakis* eggs or in earthworms which act as transport hosts for the eggs, with the life cycle continuing when the eggs or earthworms are ingested by the final host. In the present study, the first four cases occurred 10 to 28 days after the completion of a course of the in-feed anthelmintic flubendazole and wet preparations from the caeca of these birds did not detect any *H. gallinarum*. The fifth case occurred 35 days after anthelmintic treatment and large numbers of adult *Heterakis* were visible in the caeca. The sixth and final case occurred eight days later, by which time the birds were receiving a second course of in-feed anthelmintic, and *Heterakis* eggs and adults worms were also found in this bird.

It is evident that histomoniasis can occur in pheasants in the absence of either caecal lesions or obvious burdens of caecal nematodes, and that despite the widespread use for many years of dimetridazole in the feed of young gamebirds, the organisms are still present in earthworms in the environment and can cause disease in unmedicated birds. If dimetridazole is withdrawn from use in the future (Anon 1995), histomoniasis may reappear as a significant problem in young pheasants and partridges.

Reproductive tract disorders

Disorders of the female reproductive tract (egg yolk peritonitis, impaction of the oviduct, prolapse of the oviduct, or combinations of these conditions) resulted in the deaths of 43 birds, approximately 1 per cent of all the female pheasants placed. These deaths occurred at various times after the onset of egg production, and accounted for 27.7 per cent of the carcasses submitted from the breeding pens. Bacterial cultures were made from the abdominal cavity of some of the birds with egg yolk peritonitis and a variety of bacteria were isolated, predominantly *E. coli* but also *Pasteurella* species and *Yersinia pseudotuberculosis*. Similar reproductive tract disorders are common in domestic fowl, turkeys, and ducks, and are caused by several factors including excess bodyweight, the production of large eggs, vent pecking, ascending infections of the reproductive tract, physical upsets of the birds, *M. gallisepticum* infection, and other reproductive stressors (Goederham 1993, Stuart 1993, Pattison 1993). The causes in pheasants are likely to be similar, and if losses from reproductive tract disorders become excessive in a flock of breeding pheasants, the role of these predisposing factors should be investigated.

Trauma

Trauma was considered to be the cause of death of 35 birds (28 females and seven males), making up 22.6 per cent of all deaths. There was a difference between the sexes, with trauma causing 43.7 per cent of all male deaths compared with 20.1 per cent of all female deaths. The traumatic injuries in the males were mostly caused by head pecking, and in females the commonest traumatic injury was loss of feathers, excoriation of the skin, and subsequent cellulitis along the back, resulting from injuries acquired during treading (matting) and subsequent cannibalism. The majority of the treading injuries occurred in the third or fourth production periods. Losses from trauma accounted for approximately 1 per cent of all the pheasants placed in 1996 and 1997. Wise (1993) commented that such losses could reach 20 per cent. The relatively low incidence of such disorders may have been due to the use of plastic spectacles, the blunting of spurs of the male birds, and to the birds being kept in small groups at a low stocking density. The use of canvas saddles on the hens can reduce treading injuries (Wise 1993), as does the provision of adequate cover. Conversely, losses from treading injuries can increase if the above factors are not dealt with, especially in wet, muddy conditions when more feathers may be removed and greater excoriation of the back may occur during mating.

Lymphomatous tumours

Lymphomatous tumours were found in eight birds. Five of the birds had enlarged livers, usually pale but sometimes green or reddish, with paler focal lesions varying from military lesions, 0.5 mm in diameter, to large protruding nodules 4 cm in diameter. A sixth bird had a very enlarged pale liver with no focal lesions. Four of the birds with affected livers also had enlarged pale spleens, sometimes with a marbled appearance. In the remaining four birds the spleens were grossly normal. Two birds had enlarged thymus glands, with individual lobes reaching 4 cm in diameter, and two birds had pale nodules up to 8 mm in diameter on the mucosa of the duodenum. One bird had pale foci in the kidneys in addition to other lesions, and another bird had pale areas in the pancreas and caeca, and pale protruding masses up to 8 mm in diameter on
the hard palate. In one bird, the only abnormality detected consisted of multiple pale nodules, 2 to 3 cm in diameter, some of which had ulcerated, on the mucosa and serosa of the proventriculus. Another bird, the only male with lymphomatous tumours, had marked nodular thickening of the eyelids and subcutaneous oedema of the unfeathered areas of the legs, resulting in thickening of the legs and distortion of the scales.

Histopathological examination of the affected tissues revealed that the tumours were composed of cells of the lymphoid series. In some birds, autolysis prevented further identification of the cell types present, but in most birds the tumours were made up of a pleomorphic population of lymphoblasts and small, medium and large lymphocytes. Macrophages and reticulum cells were also observed in some of the neoplastic masses. Tumours of the livers were usually perivascular and coalescing in distribution. A polymerase chain reaction (PCR) test for reticuloendotheliosis virus (REV) was inconclusive in tumours from three birds, but the liver and spleen of a fourth affected bird were positive for REV. Tumours from four birds were tested by PCR for evidence of Marek's disease antigen, with negative results.

Viruses-induced tumours of cells of the lymphoid series have been described in several species of birds of the order Galliformes, an order which includes the domestic fowl, turkey, and various species of pheasant, partridge, quail, grouse, ptermajon, capercaillie, peafowl and guinea fowl. The viruses associated with such tumours in domestic fowl belong to one of three different groups: Marek's disease virus (MDV), an oncogenic herpesvirus; avian leucosis virus (ALV), an oncogenic retrovirus; and REV, another oncogenic retrovirus (Witter 1997). In turkeys such tumours have been associated with REV (McDouggall and others 1978), MDV (Malkinson and others 1996) and with another oncogenic retrovirus named lymphoproliferative disease virus (LPDV) (Biggs and others 1978). Lymphomatous tumours of uncertain aetiology have also been recorded in Japanese quail (Coturnix coturnix japonica) (Wight 1963, Carlson and others 1974, Sait and others 1976).

Tumours induced in domestic fowl and turkeys by MDV are composed of a pleomorphic mixture of lymphoblasts, small medium and large lymphocytes, and reticulum cells. Lymphoid tumours induced in domestic fowl by ALV are made up of uniform lymphoblasts (Calnek and Witter 1991). Infections in domestic fowl with REV have been associated with an acute reticulum cell neoplasia, in which the tumour cells are large vesicular, reticulum or reticuloendothelial cells (Payne 1992), and bursal and non-bursal lymphomas comprising a homogeneous population of immature lymphoreticular cells (Witter and others 1986). Tumours in turkeys with REV also consist of uniform lymphoblastoid cells with large vesicular nuclei (McDouggall and others 1978), whereas in turkeys with LPDV, the neoplastic cells are similar to those of Marek's disease in the domestic fowl, consisting of lymphocytes, lymphoblasts and reticulum cells (Biggs and others 1978).

Naturally occurring lymphomatous conditions have also been recorded in pheasants, although their aetiology has not always been known. For example, tumours with a gross and microscopical appearance similar to those of Marek's disease in chickens have been described in pheasants (Jungherr 1999), but MDV has not been demonstrated in naturally occurring lymphomatous conditions of pheasants. Certain strains of ALV (subgroup F, subgroup G, and unclassified endogenous viruses) have been identified in some species of pheasant (Payne 1992), and embryo fibroblast cultures from ring-necked pheasants are susceptible experimentally to infection with ALV of subgroups A and E (Payne 1992), but there are no reports of naturally occurring lymphomatous tumours associated with ALV in pheasants. Lymphomatous tumours have, however, been recorded in pheasants with REV infection (Dren and others 1983) in which nodular tumours were present in the tissues of the head and/or mouth, with occasional nodular or diffuse infiltration of the wall of the crop, liver, spleen, kidney, lung and muscle. The histological appearance of the tumours in these pheasants with REV was similar to that of REV infection in chickens and turkeys, with infiltrating cells described as slightly pleomorphic lymphoblastoid cells.

The aetiology of the tumours observed in the pheasants in this study is unclear. Although REV infection has been described in pheasants and REV was demonstrated in the tissues of one of the birds, the microscopical appearance of the tumours differed from that previously described for REV in chickens, turkeys and pheasants. Witter (1997) advised caution when interpreting PCR results, because the presence of REV in a tumour does not necessarily establish a diagnosis, owing to the fact that REV can replicate in lymphomas caused by other viruses such as MDV. More work is required to establish the aetiology of such tumours in pheasants, including efforts to exclude the presence of viruses such as MDV, REV, LPDV and ALV in affected flocks.

**Other causes of mortality**

In 20 birds, the cause of death or culling was not determined or did not fall into the above categories. These less common causes of death or culling included nephrosis, fatty liver, cholangioma, impaction of the oesophagus and crop, and purulent arthritis of the foot.

**Conclusions**

There is a significant risk that current methods of pheasant breeding could jeopardise the health and welfare of pheasants. Catching up semi-wild pheasants from different sites in the spring and confining them in large groups and at high stock densities is potentially stressful and could facilitate the spread of infectious diseases among them. Breeding pens with grass floors and netted sides and the roof can allow access by wild birds and their droppings, and expose the pheasants to numerous infectious agents. The hazards of infectious disease are exacerbated by the small number of medicinal products licensed for use in adult pheasants, and the difficulties which can sometimes be encountered in administering medications to the birds in the breeding pens. The prolonged period of breeding also increases the risk of problems associated with the reproduction of lymphomatous tumours which are associated with mating and with aggression between males.

The results presented here provide some figures against which other similar breeding flocks can be compared. The study has shown that well recognised causes of high mortality, such as marble spleen disease and pheasant coronavirus-associated nephritis, can become endemic on a site and cause only low grade mortality, and it has confirmed that methods of controlling mycoplasmosis are not fully effective. The importance of helminthiasis and the necessity for regular anthelmintic treatment was also confirmed. Two unexpected causes of multiple deaths were identified, histomoniasis and lymphomatous tumours. The occurrence of histomoniasis in adult pheasants serves as a warning of the risks should routine antiprotozoal medication of young pheasants be discontinued. In this flock lymphomatous tumours caused only low-grade mortality but, depending on the aetiology of the tumours, there is potential for them to cause increased mortality in pheasants in the future.

Approximately half of all the deaths were the result of problems associated with the reproductive tract or trauma, including trauma caused by fighting between males and injuries inflicted on the females by the males during mating. Any future changes to the management of breeding pheas-
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References


ANON (1995) FC barn use of dimetridazole in food animals. Veterinary Record 137, 230


BYGRAVE, A. C. & PATTISON, M. (1973) Marble spleen disease in pheasants (Phasianus colchicus) Veterinary Record 92, 534-535


KEYMER, I. F. (1961) Infectious sinusitis of pheasants and partridges. Veterinary Record 73, 1034-1038


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Abstracts

Acute toxoplasmosis after renal transplants in three cats and a dog

Three cats and a dog became ill three to six weeks after having received a renal transplant to stage end-stage renal failure. One of the cats died before it could be treated, and toxoplasma cysts were found in sections of the renal allograft, and tachyzoites were found in other organs. The other animals died despite treatment and protosporozo cysts and tachyzoites were identified in other organs but not in the allografts, suggesting that a latent infection had been reactivated as a result of immunosuppression and led to disseminated toxoplasmosis.


Cytogenetic study of in vitro-derived bovine embryos

A total of 176 early bovine embryos (two to 16 blastomeres) were analysed cytogenetically. The embryos were produced from immature oocytes matured in vitro and fertilised by sperm prepared by the Percoll density gradient method. Slides were prepared by an ‘air-drying’ technique and the chromosomal complement was determined by Giemsa staining. Metaphase plates were found in 100 of them, but the others had only interphase nuclei. Of these 100, 18 had chromosomal abnormalities; eight were haploid, two were aneuploid, and eight were polyploid.

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