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CHICK NUTRITION AND MORTALITY IN CAPTIVE WILLOW PTARMIGAN (LAGOPUS L. LAGOPUS)

By

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HANSSEN, INGOLF and JOHN NESS: *Chick nutrition and mortality in captive willow ptarmigan (Lagopus l. lagopus).* Acta vet. scand. 1982, *23*, 456—465. — Willow ptarmigan chicks were reared during 8 years on concentrates supplemented with blueberry plants. Mortality during the first 3 weeks after hatching ranged between 33 and 65 %, and was mainly caused by enteritis and digestive tract obstructions. The annual variations in chick survival seemed to be caused by the variations in plant phenology. The survival was highest when spring and blueberry plant development was late, and lowest when spring was early and warm, leading to early lignification of blueberry plant leaves.

willow ptarmigan; nutrition; mortality.

Rearing willow ptarmigan in capivity was attempted in Norway for the first time already in 1937 (*Høst* 1938, *Holt* 1946). The investigators found it extremely difficult to get the captive chicks through their first weeks. The few that survived were fed natural feeds like insects and blueberry plants (Vaccinium myrtillus). At the Nature Conservancy at Banchory in Scotland large scale rearing of red grouse (Lagopus lagopus scoticus) has been done for more than 20 years. The chick feed used in this stock has been different brands of turkey, pheasant and partridge starters, supplemented with heather (Calluna vulgaris). During this period it has been noted that chicks hatched from eggs of captive birds show a fairly constant survival of 50—80 %, while survival in captivity of chicks hatched from eggs of wild birds is very variable from one year to another, and parallels the survival of the wild chicks on the moor (*Moss* 1969).

Since 1972 willow ptarmigan have been reared for experimental purposes at the University of Tromsø. The aim of the present article is to present the results from our chick rearing obtained up to 1980.

MATERIAL AND METHODS

Breeding ptarmigan

Eggs were derived from both wild and captive ptarmigan hens. Eggs from wild birds were usually found by flushing the incubating hen from her nest, and the eggs were brought to the laboratory in insulated bags with a temperature of 35°C. Eggs from captive birds were derived from our own ptarmigan stock. These birds were kept as described by *Hanssen et al.* (1982).

In 1973 about equal numbers of the eggs were incubated by bantam hens and in machines, while later on all eggs were incubated and hatched in machines (*Hanssen et al.* 1982). The chicks were kept and dried in the hatcher for 12—24 h, before they were taken out, weighed and tagged.

Rearing of chicks

The chicks were brought into aluminium brooder houses of the same type as described by *Moss* (1969). The floor area was 1.2 m^2, and made by 0.5″ wire mesh. The houses were placed on blueberry plant/grass ground. Heat, 35°C at chick level, was provided by two 250 W infra-red heat lamps. Feed was placed in many small low trays around in the brooder house. The composition and analysis of our chick diet are given in Table 1. There were, however, modifications from one year to another (Table 2). The chick diet was supplemented with hand picked bundles of blueberry plants, from which unripe berries were removed to prevent oesophagus obstruction (*Hanssen & Parker* 1976). Water was provided in conventional chick water-fountains. These were filled with stones during the first week to prevent drowning. Granite and quartz grit was also given. Depending on the weather, the chicks were let out into a 3 m^2 run when they were 2—5 days old. Each brooder unit could hold up to 40 chicks. To obtain a good hygienic standard, feed was replenished and water fountains were washed once a day. Once a week the brooder houses were moved to a fresh place.

Table 1. Composition and analysis of artificial ptarmigan chick feed (see also Table 2).

Ingredients	% fresh wt.	Analysis	% dry basis
Herring meal	10.0	Dry matter	89.6
Soya meal (extracted)	10.0	Oil	5.5
Maize	25.0	Crude protein	22.0
Wheat	25.0	Crude fibre	3.3
Wheat bran	16.9	Ca	2.0
Brewers yeast	2.0	P	1.0
Limestone	1.5	Mg	0.2
Calcium phosphate	2.0	K	0.8
Skimmed milk powder	2.0	Na	0.3
Blood meal	2.0	Cl	0.3
Whey powder	2.0		
Vitamin premix[1]	1.0		
Trace mineral premix[2]	0.4		
Ascorbic acid	0.2		

[1] Supplies pr. kg: 7500 I.U. vit. A, 1480 I.U. vit. D_3, 250 mg vit. E, 25 mg vit. B_1, 150 mg vit. B_2, 45 mg vit. B_6, 55 mg Ca-D-pantothenate, 550 mg Niacin, 10 mg Folic acid, 3525 mg Choline chlorid, 0.45 mg Biotin, 0.01 mg B_{12}, 10 mg Vit. K_3, 550 mg Inositol, 25 mg para-amino-benzoic acid, 2265 mg Ascorbic acid, 75 mg Etoxiquin.

[2] Supplies pr. kg: 172 mg Fe, 228 mg Mn, 200 mg Zn, 57.2 mg Cu, 4.4 mg Co, 8 mg I.

Post mortem examinations of dead chicks

Date of death and body weight of chicks that died were recorded, and the corpses were placed in plastic bags in a refrigator or freezer till necropsy could be done. The necropsy included histological, bacteriological and parasitological investigations. Except in 1977, 50—90 % of the chicks that died each year were necropsied.

RESULTS

The mortality during the first 3 weeks after hatching is shown in Fig. 1. The mortality for the different years varied between 33 and 65 %, and was always high during the first week after hatching. In Table 3 the causes of deaths are shown. Enteritis, digestive tract obstructions and congenital infirmity were the most frequently occurring disorders. In the following a brief description is given of the different mortality causes.

Table 2. Feed and water additives, and modifications of diet 1973—1980.

Year	Barley and oats in the diet	Oxytetracycline[1]	Amprolium[2]	Ascorbic acid	Flavonoids
1973 and 1974	10 % each	50 mg/l drinking water to chicks in flocks with diarrhoea	0.012 % in the drinking water on day 8—12 after hatching	—	—
1975 and 1976	10 % each	50 mg/l drinking water during first week	0.012 % in the drinking water on day 8—12 after hatching	—	—
1977	10 % each	50 mg/l drinking water during first week	0.012 % in the drinking water on day 8—12 and day 18—22 after hatching	—	—
1978	10 % each	—	0.012 % in the drinking water on day 8—12 and day 18—22 after hatching	0.2 % in the drinking water	—
1979	—	—	0.012 % in the drinking water on day 8—12 and day 18—22 after hatching	0.2 % in the food	—
1980	—	—	0.012 % in the drinking water on day 8—12 and day 18—22 after hatching	0.2 % in the food	0.1 % rutin and quercetin in the feed

[1] Terramycin vet. "Pfizer".
[2] Amprolsol vet. "MSD".

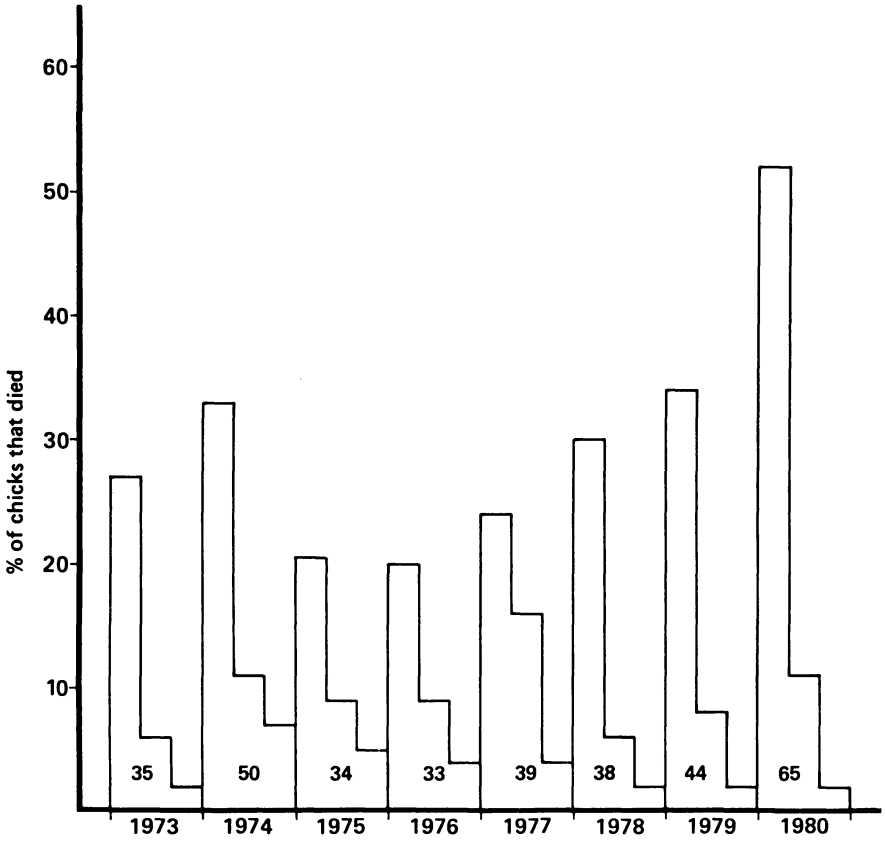

Figure 1. Chick mortality in the period 1973—1980. The left part of each column unit represents the first week, the middle part the second week, and the right part the third week after hatching. The number within each column unit is the total mortality (%) during the first 3 weeks after hatching.

Enteritis occurred as early as 2—3 days after hatching. Chicks suffering from enteritis usually had watery feces and gradually became lethargic before death occurred. Feces commonly adhered to the feathers around the anus of these chicks, and if not removed, might build up to a plug in the cloaca resulting in gut obstruction. The main necropsy findings were emaciation and scarce, gaseous and partly hemorrhagic gut contents. The gut mucous membranes were more or less slimy, hyperemic and edematous. The gut segment most heavily affected was the lower part of the small intestine. Bacteriological

Table 3. The causes of death in willow ptarmigan chicks during the first 3 weeks after hatching.

Year	Initial no. of chicks	Mortality, %	Mortality (%) according to cause						
			Enteritis	Digestive tract obstructions	Congenital infirmity	Overconsumption of grit	Pneumonia	Coccidiosis	Other causes
1973	365	35	12	4	8	—	1	4	6
1974	368	50	18	5	8	—	7	4	8
1975	496	34	16	6	1	1	1	3	6
1976	369	33	10	1	12	—	—	—	8
1977*	406	39	—	—	—	—	—	—	—
1978	479	38	13	10	3	2	—	—	10
1979	581	44	12	13	6	—	—	—	13
1980	326	65	16	29	5	6	1	—	8

* Only 21 % of the dead chicks were necropsied.

examinations of gut contents showed high numbers of coliforms and enterococci of different types. E. coli strains, isolated from chicks dead from enteritis were serotyped and tested for thermolabile enterotoxin production*, but none of them were classified as enterotoxic. Occasionally coliforms or enterococci were isolated from the liver of chicks. In septicemia caused by enterococci, petecchial hemorrhages in the myocardium and fibrinous exudate in the lungs and pleural cavity were regularly observed. In septicemia caused by coliforms, hyperemia in the liver and kidneys was the only finding. Administration of both oxytetracyclin and ascorbic acid (Table 2) leads to reduction in numbers of coliforms (*Hanssen et al.*, unpublished). Nevertheless, the frequency of enteritis was substantial in all years (Table 3).

Digestive tract obstruction. Although pebbles and sand were available, many chicks died every year because of lack of grit in their gizzards. This led to impaired gizzard function, and many chicks died with whole leaves of blueberry plants stuffed into the duodenum.

From the beginning of July and onwards chicks were often found dead with the intestine obstructed by blueberry leaves, though they had normal amounts of grit in the gizzard. The reason for this obviously was that the content of both dry matter

* Performed at Statens Seruminstitut, Copenhagen, Denmark.

and fibers increases during the summer, and at a certain stage of development the gizzards of very young chicks became unable to grind the bluberry leaves.

Many ptarmigan chicks also died because they ate grass, small pieces of wood and other foreign bodies, which became stuck in the crop or further caudally in the digestive tract. Among chicks with intestinal obstruction it was quite common that the atria of the heart showed blood congestion and that the atrial wall was ruptured and the pericardial sac was filled with blood.

In the years when barley and oates were included in the diet, it was noticed that many of the chicks that died had large quantities of small rigid husks in their gizzard.

Congenital infirmity. The definition of this condition poses problems. It is easy to agree that a chick unable to get out of the shell on its own, or falling on its back, or spraddling its legs, is weak. However, there is a continuous transition from such manifestations via more or less drowsy to the fully vital chicks. In the present study chicks with curly toes, or being exceptionally small, or presenting no other necropsy findings than an empty digestive tract 2—3 days after hatching, also were classified as having died due to congenital infirmity.

Overconsumption of grit. While some chicks died from digestive tract obstruction, due to lack of grit in the gizzard, other chicks died because of overconsumption of grit. Some of these chicks had too many or too large pebbles in the gizzard, while others had the entire digestive tract filled with sand. This no doubt also predisposed for enteritis.

Pneumonia was once introduced in 1974 with wild chicks brought from the field to our station in bags heated with warm, mouldy barley (*Hanssen* 1975). The few other cases of pneumonia that occurred, could be traced to fungi growing in the brooder houses as a result of feed and water spill. The typical pathoanatomical picture was small greyish-white nodules in the lungs and airsacs.

Coccidiosis occurred from day 10. If not prevented by drugs, this disease could bring heavy losses. The diseases was characterized by a sudden onset. Apparently healthy chicks fell into convulsions and died within a few hours. Post mortem

examinations showed most often a well nourished body and a small intestine stuffed with chyme. There was no macroscopical inflammatory reaction in the intestine, but scrapings taken from the small intestinal and caecal mucosa showed large numbers of oocycts. The most common species was Eimeria fanthami, but Eimeria brinkmanni was also observed.

Other causes of death. In Table 3 there is a column in which deaths due to other causes are presented. This category includes chicks that drowned, that died due to trauma, that were unfit for necropsy because of heavy cadaverosis, and also a few for which the cause of death could not be found. Each year some chicks died because the yolk sac was infected by coliform bacteria, eventually leading to septicemia. Cannibalism also occurred, and nephritis and abnormal gizzard lining were occasionally observed.

DISCUSSION

The chicks were particularly susceptible to enteritis during the first week (Fig. 1 and Table 3). Their intestine seemed to be very sensitive even to traditionally nonpathogenic bacteria, and although administration of antibiotics, ascorbic acid and blueberry plants clearly effect both their gut microflora and improve their growth rate (*Hanssen et al.* 1979). still many chicks died from enteritis.

The other main cause of death was digestive tract obstruction (Table 3). Blueberry plant is a natural feed item for willow ptarmigan chicks, and supplement of this plant obviously reduced the psychological stress in the flock and improved the well-being of the chicks. It also gives bulk to the total ration, and contains antimicrobial substances (*Stangeland* 1980). However, the blueberry plants in Tromsø normally are too fibrous for the very young chicks after the beginning of July.

In 1980 blueberry plant development was extremely early (*Hanssen et al.*, unpublished) and had therefore to be deleted from the ration for most of the chicks during the first week after hatching. This led to many deaths due to intake of grass and other material obstructing the digestive tract. It is our experience that successful rearing of chicks without natural feed supplement is possible only if ascorbic acid and antibiotics are administered in the feed or drinking water, if the chicks are

kept in small groups with plenty of shelter, and if they are prevented from eating sand, stones, twigs and grass (*Hanssen et al.* 1979). However, at the time when blueberry plants are introduced, one has to be careful because overconsumption leading to digestive tract obstruction is then very likely to occur.

Moss (1969) reported that the survival of red grouse chicks hatched from eggs of wild birds and brought up in captivity varied a great deal from one year to another, while the survival of chicks from eggs of captive hens was more constant. He suggested as a reason annual variations in egg quality of wild red grouse. Detailed analysis of eggs from wild willow ptarmigan revealed significant annual differences, but these differences could not be related to observed annual variations in chick survival (*Hanssen et al.*, to be published). A comparison of eggs from wild and captive birds did neither demonstrate marked differences. Eggs from wild birds tended to hatch better than eggs from captive ones, which was most likely due to the fact that the former were started naturally, while the latter were incubated in machines during the whole period (*Hanssen et al.* 1982). Furthermore, chicks from eggs of wild birds grow better on a vitamin C deficient diet than chicks from captive birds (*Hanssen et al.* 1979). Nevertheless, the chick survival in our rearing has been the same for chicks from eggs of wild and captive birds (*Hanssen et al.* 1982). In 1976 mortality due to congenital infirmity was higher than in the other years (Table 3). The reason was probably that the humidity in the incubator was lower that year (*Hanssen et al.* 1982). Otherwise, we have seen that the mortality every year has varied considerably from one brooder unit to another. The reasons for these variations are obscure, but we suggest that only minor variations in flock composition and environmental conditions may influence the thriving of the chicks. The annual variations in mortality observed in the present work (Fig. 1, Table 3) can be related to the digestibility of the blueberry leaves. Mortality was lowest when spring was cold and late, leading to low fiber contents in the blueberry leaves at the peak hatching period, and highest in 1980 when blueberry plants were extremely dry and fibrous in the critical period.

REFERENCES

Hanssen, I.: Pulmonary phycomycosis in captive rock ptarmigan (Lagopus mutus) and willow ptarmigan (Lagopus l. lagopus) chicks. Acta vet. scand. 1975, *16*, 134—136.

Hanssen, I. & H. Parker: Throat obstruction as a mortality factor among willow ptarmigan chicks. Wilson Bull. 1976, *88*, 353.

Hanssen, I., H. J. Grav, J. B. Steen & H. Lysnes: Vitamin C deficiency in growing willow ptarmigan (Lagopus lagopus lagopus). J. Nutr. 1979, *109*, 2260—2276.

Hanssen, I., J. Ness & J. B. Steen: Parental nutrition and chick production in captive willow ptarmigan (Lagopus l. lagopus). 1982 (to be published).

Holt, E.: Forsøk med oppdrett av ryper. (Experiments on rearing willow ptarmigan). Norges Jeger- og Fiskerforb. Tidsskr. 1946, *75*, (8): 19—26, (9—10): 17—29.

Høst, P.: Forsøk med oppdrett av ryper. (Experiments on rearing willow ptarmigan). Norges Jeger- og Fiskerforb. Tidsskr. 1938, *67*, 4—15; 31—43; 137—144.

Moss, R.: Rearing red grouse and ptarmigan in captivity. Avicult. Mag. 1969, *75*, 256—261.

Moss, R. & I. Hanssen: Grouse nutrition. Nutr. Abst. Rev. — Ser. B, 1980, *50*, 555—567.

Stangeland, K.: Vannløselige organiske syrer og antimikrobielle stoffer i blader av Vaccinium myrtillus L. (Water soluble organic acids and antimicrobial substances in leaves from Vaccinium myrtillus L.). B. Sci. thesis, Farmasøytisk institutt, Universitetet i Oslo, 1980.

SAMMENDRAG

Ernæring og mortalitet hos lirypekyllinger i fangenskap.

I 8 år er lirypekyllinger oppdrettet på kraftfór og blåbærlyng. Mortaliteten i løpet av de 3 første ukene etter klekking varierte mellom 33 og 65 %. De viktigste dødsårsakene var enteritt og obstipasjoner i fordøyelsestraktus. Variasjonene i kyllingoverlevelse fra år til år syntes hovedsakelig å skyldes årsvariasjonene i utviklingen av blåbærplantene. Overlevelsen var best i år da våren og blåbærlyngutviklingen var sein, og dårligst i år da våren og lignifiseringen av blåbærlyngen var tidlig.

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