Multiannual fluctuations in willow ptarmigan Lagopus l. lagopus - does the genetic variation of nesting females enhance the effect of predation?

Authors: Rørvik, Kjell-Arne, Pedersen, Hans Christian, Olli, Jan, Gjøen, Hans Magnus, and Steen, Johan B.

Source: Wildlife Biology, 5(3) : 137-145

Published By: Nordic Board for Wildlife Research

URL: https://doi.org/10.2981/wlb.1999.018

Multiannual fluctuations in willow ptarmigan *Lagopus l. lagopus* - does the genetic variation of nesting females enhance the effect of predation?

Kjell-Arne Rørvik, Hans Christian Pedersen, Jan Olli, Hans Magnus Gjøen & Johan B. Steen

Rørvik, K-A., Pedersen, H.C., Olli, J. Gjøen, H.M. & Steen, J.B. 1999: Multiannual fluctuations in willow ptarmigan *Lagopus l. lagopus* - does the genetic variation of nesting females enhance the effect of predation? - Wildl. Biol. 5: 137-145.

Willow ptarmigan *Lagopus l. lagopus* is a popular game bird which fluctuates in abundance. The causes of these fluctuations, however, remain controversial, but several studies have emphasised the effect of predation. Predation not only reduces the number of breeding birds, but does it in such a way that genetic variation among chicks at hatching becomes reduced, causing reduced viability and increased mortality among chicks. We present an extended predation hypothesis in which the multiannually fluctuating population dynamics of the willow ptarmigan are better explained by a model including both predation and genetic variation of territorial nesting females than by predation alone. A simple model including the heterozygosity of nesting territorial females and the percentage of females suffering egg predation explained 95% of the observed fluctuations in chick production on an inland study area during five years, whereas predation alone only explained 72%. The data may suggest a non-additive relationship between predation and genetic variation of nesting females which enhance the effect of predation. Observed and calculated chick production per two adults deviated on average by only 0.38 chicks. In another inland population, showing multiannual fluctuations for almost 20 years, observed and calculated chick production deviated on average by 0.58 chicks, and the model explained 61% of observed fluctuations in chick production, whereas predation alone only explained 28%. In an island population, however, the full model explained 45% of observed fluctuations in chick production. This was about the same as predation alone (44%). It is discussed whether the better fit of the full model than the model including predation alone between observed and calculated chick production obtained in the two inland populations in contrast to the island population, may be caused by the different predator communities.

Key words: fluctuations, genetics, Lagopus lagopus, predation, willow ptarmigan

Kjell-Arne Rørvik, Department of Biology, Division of General Genetics, University of Oslo, P.O. Box 1031 Blindern, N-0315 Oslo 3, Norway*
Hans Christian Pedersen, Norwegian Institute for Nature Research, Division of Terrestrial Ecology, Tungasletta 2, N-7485 Trondheim, Norway
*Jan Olli** & Hans Magnus Gjøen, AKVAFORSK, Institute of Aquaculture Research Ltd., P.O. Box 5010, N-1432 Ås, Norway*
Johan B. Steen, Department of Biology, Division of General Physiology, University of Oslo, P.O. Box 1051 Blindern, N-0315 Oslo 3, Norway

Present addresses:

** AKVAFORSK, Institute of Aquaculture Research. P.O. Box 5010, N-1432 Ås, Norway - e-mail: kjell-arne.rorvik@akvaforsk.nlh.no*
***BioMar Ltd., N-8430 Myre, Norway*

Received 13 June 1997, accepted 5 May 1999

Associate Editor: Peter J. Hudson

Willow ptarmigan *Lagopus l. lagopus* is widely distributed over arctic and subarctic Eurasia and North America where most of the populations exhibit cyclic fluctuations in abundance. The causes of these fluctuations remain controversial (Lindström 1994). In Scandinavia, willow ptarmigan undergo 3-4 year cyclic fluctuations in population density (Myrberget 1972, Steen, Stenseth, Myrberget & Marcström 1988a). Over 40 years ago, Hagen (1952) related the ptarmigan fluctuations in Scandinavia to changes in predation pressure and noted that chick production varied inversely with the density of the small rodent population and thus formulated "the alternative prey hypothesis". This hypothesis states that eggs and chicks of tetraonids are alternative prey for generalist predators which mainly prey on small rodents. When small rodents are abundant, these predators eat fewer eggs and chicks; when rodents are scarce, the predators switch to eggs and chicks of tetraonids. Several studies have supported this hypothesis and emphasised the effect of egg predation in spring on autumn numbers of juvenile willow ptarmigan in Scandinavia (Myrberget 1972, 1988, Marcström & Höglund 1980). However, fluctuations in predation pressure alone can only partly explain the annual fluctuations in chick production. Steen et al. (1988a), modelled the fluctuating variations in chick production of a Swedish inland population (Lövhögen) and of a Norwegian island population (Tranøy) and found biotic variables (an index of predation) to be more important than abiotic variables (weather). In the two populations, predation explained only 25 and 30% of the annual variation in autumn number of juveniles, respectively. Consequently, other variables are probably involved, either separately or in relation to predation. A new approach based on reanalyses of the Tranøy data was suggested by Steen & Erikstad (1996) who claimed that first year winter mortality was equally or even more important than survival of eggs or chicks in affecting growth rate in the willow ptarmigan population.

The importance of genetic components in explaining multiannual fluctuations in tetraonids has been discussed by several authors (e.g. Watson & Moss 1979, Page & Bergerud 1984, Hannon 1988, Moss & Watson 1991). Although not thoroughly tested, some studies have supported Chitty's Polymorphic Behavioural Hypothesis based on small rodent studies (Chitty 1967), suggesting that fluctuations are driven by interactions between aggressive and non-aggressive genotypes. However, in our study population, we did not find any significant difference among years in the average heterozygosity of either juvenile or adult territorial males or females (Rørvik, Pedersen & Steen 1990), even though mean territory size during the same period varied 2.3 fold (Pedersen 1984, H.C. Pedersen, unpubl. data). Following a recent reanalysis of data from a fluctuating red grouse *Lagopus l. scoticus* population, it is suggested that changes in behaviour that generate fluctuations may result from kin selection; i.e. kin show less aggressive behaviour than non kin (Moss & Watson, 1991, Watson, Moss, Parr, Mountford & Rothery 1994). One important assumption for the kin-selection model is that juvenile males establish territories close to their fathers' territories and thereby reduce the hostility between close kin (Moss & Watson 1991, Watson et al. 1994). This has been observed in red grouse (Lance 1978, Moss & Watson 1991, Watson et al. 1994), but not in our study population (Pedersen, Steen & Andersen 1983, H.C. Pedersen, unpubl. data). Thus, in our study population, it seems that neither the Chitty hypothesis nor the kin-selection model could explain the observed annual changes in chick production.

Analyses of single genes have implied changes in gene and genotype frequencies in blue grouse *Dendragapus obscurus* and red grouse in response to changed population density (Redfield 1973, Henderson 1977). Gyllensten (1985) expanded the scope of earlier studies in birds by monitoring changes in gene frequencies in six polymorphic genes. He found genetic variations in willow ptarmigan from five Scan-

dinavian populations. However, during a five-year genetic study of willow ptarmigan in Norway and Sweden, most gene diversity (97.6%) was found within populations within years, whereas only 1.5% and 0.9% were attributed to temporal and spatial differences, respectively (Rørvik & Steen 1989).

In a survival study of free-living ptarmigan chicks in our study area, monitoring changes in eight polymorphic genes, the impact of ambient temperature, predation and genetic variation of the chicks was evaluated (Rørvik, Pedersen & Steen 1999). Consistent with several earlier survival studies in Scandinavian willow ptarmigan (Holt 1953, Myrberget 1986, Steen, Andersen, Sæbø, Pedersen & Erikstad 1988b) and in other *Lagopus lagopus* subspecies (Jenkins, Watson & Miller 1963, Bergerud 1970), ambient temperature in the chick-rearing season showed no association with survival of the chicks. Observation of a significant negative association between genetic relatedness of mates and chick survival might imply that more chicks die in their first week of life when genetic relatedness of mates is high (i.e. low chick heterozygosity at hatching). Hence, newly hatched chicks with low heterozygosity may have reduced viability and, therefore, might suffer higher mortality due to biotic/abiotic conditions, matching the general view that inbred individuals with low genetic variation generate reduced fitness (Falconer 1981). Due to the observation of a highly significant negative association between the proportion of surviving chicks with low heterozygosity and predation pressure, it is proposed that genetic variation significantly affects the viability of ptarmigan chicks, but that predation is the proximate cause of death. Predation was related also to the genetics of nesting females (Rørvik et al. 1999). More predation among nesting females with high than low heterozygosity reduces genetic variation in the breeding population. Reduced genetic variation among parents causes reduced genetic variation among chicks. Hence, in agreement with several earlier studies, predation was related both to nesting females and to survival of the chicks. However, our study extends these findings by suggesting a relationship between predation on one side and genetic constitution on the other, and that predation among nesting females, by reducing genetic variation among chicks at hatching, enhance chick mortality.

Because predation not only reduces the number of breeding birds, but does it in such a way that genetic variation among chicks at hatching becomes reduced, causing reduced viability and increased mortality among chicks, we present an extended predation hypothesis in which the multiannually fluctuating population dynamics of the willow ptarmigan are better explained by a model including both predation and genetic variation of territorial nesting females than by predation alone.

Methods

Our data are based on observations from Gåvålia in Dovrefjell National Park in central Norway (62°17'N, 09°39'E) collected during 1981-1985, from Lövhögen (62°N, 13°E) in central Sweden collected during 1967-1983 and from Tranøy (69°N, 17°E) in northern Norway collected during 1965-1982. The Gåvålia and Lövhögen areas both are in typical inland and alpine willow ptarmigan habitats, whereas Tranøy is a 1.25 km^2 island where ptarmigan breed from sea level to the summit of the island which is 32.5 m a.s.l.

In Gåvålia, females were mostly caught on the nest with a hand net in June. Blood samples were taken from a wing vein. Genotypes were identified by means of electrophoretic separation of serum esterases (EST) in polyacrylamide gels using isoelectric focusing with carrier ampholyte (PAGE-IEF) (Rørvik 1987). Family studies have shown that eight polymorphic genes (Rørvik 1989) inherit the various forms of the enzyme.

The proportion of females suffering egg predation was recorded in all years (Rørvik et al. 1999). Chick production in the autumn was recorded by pointing dog censuses and expressed as observed number of chicks per two adults during 15-20 August. The Lövhögen and Tranøy test data are from Steen et al. (1988a). All statistics are regression analyses (F) using the SAS software package (SAS 1989). The percentage of total variation explained by the model is expressed by R^2. Both sequential sums of squares (in the SAS manuals referred to as type I SS), which depend on the sequence in which the model variables are ordered, and the partial sums of squares (in the SAS manuals referred to as type III SS), which gives the marginal effect of each variable, are checked and compared. Large difference in the sequential and partial sums of squares may suggest that the two model variables are related to each other in a non-additive way; for instance a high level of one of the variables may cause the effect of the other variable to be en-

hanced compared with what it is at low levels of the first variable. If not stated otherwise, the P-values in the regression analyses are based on the marginal effect of each variable.

Based on the observations of Rørvik et al. (1999), a significant positive relationship between heterozygosity of nesting females and predation may suggest more predation among territorial females with high than low heterozygosity. This is consistent with the fact that twice as many females with four or more heterozygous genes as females with three or fewer heterozygous genes are preyed on. It is also consistent with increased mean genetic relatedness among birds in the breeding population when females suffering predation were excluded. Hence, the heterozygosity of females was defined to be high if they had four or more heterozygous genes out of the eight studied, and chick production (C_P) in a given year (t) was calculated using the following equation:

$$C_P(t) = a\, H_F(t) + b\, P_F(t) + c \qquad (1),$$

where H_F is the percentage of territorial females with four or more heterozygous genes, P_F is the percentage of females suffering predation, and a, b and c are constants fitting the model to observed values of chick production.

Information is scarce about the proportion of females suffering predation and particularly about the heterozygosity of nesting females in bird populations. Therefore, in willow ptarmigan populations other than our study population, it was necessary to find substitutes correlated with components in equation 1. Myrberget (1985) found a negative relationship between the number of small rodents and the observed egg predation during a 23-year study at Tranøy. In Gåvålia, the number of small rodents was ranked (N) on the basis of observations as: 1) hardly any seen, 2) few seen daily and 3) many seen daily. Such a ranking has been found to agree well with more quantitative methods (Myrberget 1982), and was also used by Steen et al. (1988a). The observed proportion of females suffering predation varied negatively with the ranked number of small rodents observed in Gåvålia during 1981-1985 (see Table 1). The ranked number of small rodents explained 95% of the annual variation in the proportion of females suffering predation ($F = 52.54$, $df = 3$, $P = 0.005$). To generate a positive association with the predation pressure, in the model the proportion of females suffering predation is substituted for 1/N which is a predation index (P_I). Most females being territorial in the spring were chicks either the previous year or the year before that. Based on the observations of predation by Rørvik et al. (1999) in Gåvålia, significantly more adult than juvenile females with four or more heterozygous genes suffered predation. Further, significantly higher chick survival was observed in years with high heterozygosity expected among chicks at hatching than in years with low heterozygosity. Accordingly, the number of chicks with four or more heterozygous genes increased significantly with increasing chick production, whilst the number of chicks with three or fewer heterozygous genes decreased significantly with increasing chick production ($F = 89.91$, $P = 0.003$, $R^2 = 97\%$). Because most adult females in a given year were chicks two years before, a positive association is expected between the proportion of adult nesting females with four or more heterozygous genes and chick production two years before. Accordingly, regression analysis showed that the percentage of adult territorial females with four or more heterozygous genes was positively associated with chick production two years earlier ($F = 8.97$, $P = 0.05$, $R^2 = 75\%$). Therefore, percent females with four or more heterozygous genes in a given year is substituted by the chick production two years earlier, and chick production (C_P) in a given year (t) is calculated using the following equation:

$$C_P(t) = a\, C_P(t\text{-}2) + b\, P_I(t) + c \qquad (2),$$

where $C_P(t\text{-}2)$ is the chick production observed two years earlier, P_I is the predation index (1/N, N = ranked small rodent index) and a, b and c are constants fitting the model to observed values of chick production.

Results

Using equation 1, the regression analyses of data from Gåvålia (Table 1), for which the model included the percentage of all females with four or more heterozygous genes alone, explained only 2% of the variation in annual chick production ($F = 0.05$, $df = 3$, $P = 0.83$). When the model included the proportion of females suffering predation alone, the model explained 72% ($F = 7.82$, $df = 3$, $P = 0.07$). Finally, when including both percent territorial females with four or more heterozygous genes (H_F) and percent females suffering predation (P_F), the model explained

Table 1. Percent females suffering egg predation, ranked density of small rodents (N), percent nesting females with four or more heterozygous genes and chick production in a given year ($C_p(t)$) and two years earlier ($C_p(t-2)$) in Gåvålia, central Norway, during 1981-1985. Sample sizes are given in parentheses. See text for details.

Year	% ♀♀ suffering egg predation	N	% ♀♀ with ≥4 heterozygous genes	% adult ♀♀ with ≥4 heterozygous genes	$C_p(t)$	$C_p(t-2)$
1981	0.0	3	60.0 (10)	30.0	4.3	3.4
1982	37.5	1	50.0 (26)	30.8	0.7	1.7
1983	27.3	2	53.8 (13)	53.8	1.6	4.3
1984	18.2	2	50.0 (16)	18.8	5.3	0.7
1985	0.0	3	52.4 (21)	28.6	6.3	1.6

95% ($F = 19.44$, $df = 2$, $P = 0.05$) of the annual fluctuations in chick production during 1981-1985. The P-value (type III SS) for each individual factor in the model was 0.09 for H_F and 0.03 for P_F. Calculated values deviated on average by only 0.38 chicks per two adults from observed values these years (Fig. 1 - equation 3). We used the following equation in the calculation:

$$C_P(t) = 25.1828 - 0.1752\ P_F(t) - 0.3500\ H_F(t) \quad (3).$$

Using equation 2, the regression analyses of the data from Gåvålia (see Table 1), for which the model included chick production two years earlier alone, explained only 19% of the variation in annual chick production ($F = 0.69$, $df = 3$, $P = 0.47$). When based on predation index alone, the model explained 59% ($F = 4.35$, $df = 3$, $P = 0.13$). Thus, no single variable could significantly explain the fluctuations in chick production. The full model, including both chick production two years earlier ($C_P(t-2)$) and the predation index (P_I), explained 98% of the annual variation in chick numbers ($F = 78.32$, $df = 2$, $P = 0.01$). The P-value for each individual factor in the model was 0.02 for $C_P(t-2)$ and 0.008 for P_I. Observed and calculated values deviated on average by only 0.23 chicks these years (see Fig. 1 - equation 4). We used the following equation in the calculation:

$$C_P(t) = 10.4143 - 8.0588\ P_I(t) - 1.0587\ C_P(t-2) \quad (4).$$

There was a significant relationship between annual chick production calculated from equation 3 and equation 4 ($P = 0.01$).

Using equation 2, the regression analyses of data from Lövhögen (Table 2), for which the model included chick production two years earlier alone, explained 54% ($F = 17.28$, $df = 15$, $P = 0.0008$), whereas the model including the predation index alone explained 28% ($F = 5.83$, $df = 15$, $P = 0.03$). Thus, in the Lövhögen area, both single variables could significantly explain the multiannual fluctuations in chick production. However, the best fit between the calculated and observed chick production during 1967-1983 was found using the full model. Including both chick production two years earlier, $C_P(t-2)$, and the predation index (P_I), the model explained 61% of the annual variation in chick numbers ($F = 10.84$, $df = 14$, $P = 0.001$). The P-value for each individual factor in the model was 0.002 for

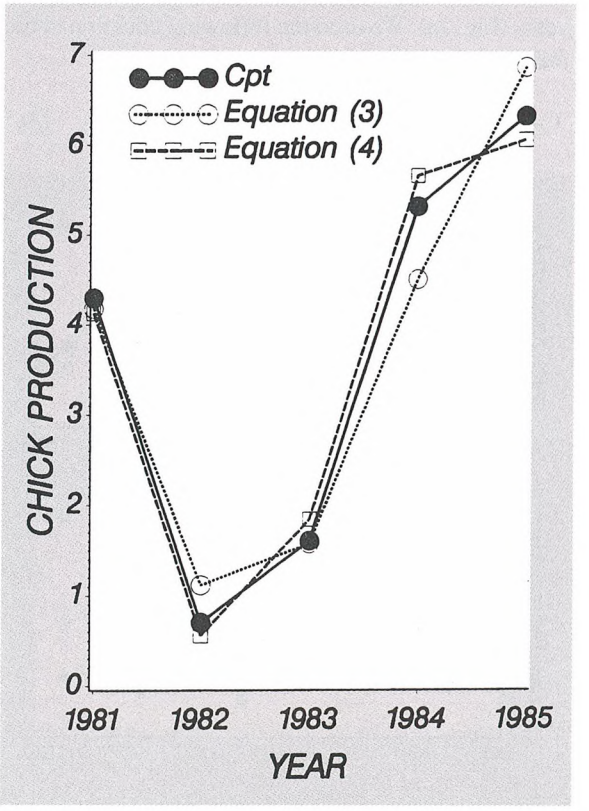

Figure 1. Observed ($C_p(t)$) and calculated (on the basis of equations 3 and 4) chick production in the inland study population at Gåvålia, central Norway, during 1981-1985. See text for details.

Table 2. Ranked density of small rodents (N), and chick production in a given year ($C_p(t)$) and two years earlier ($C_p(t\text{-}2)$) based on test data from Lövhögen, central Sweden, and Tranøy, northern Norway. See text for details.

	Lövhögen			Tranøy		
Year	N	$C_p(t)$	$C_p(t\text{-}2)$	N	$C_p(t)$	$C_p(t\text{-}2)$
1965				2	3.8	3.7
1966				3	6.8	3.8
1967	1	3.5	2.0	1	1.7	3.8
1968	2	2.2	3.5	2	3.0	6.8
1969	2	2.7	3.5	3	3.5	1.7
1970	3	2.6	2.2	3	4.0	3.0
1971	1	1.4	2.7	1	1.9	3.5
1972	2	4.2	2.6	2	3.0	4.0
1973	2	5.2	1.4	2	5.2	1.9
1974	3	3.1	4.2	3	3.0	3.0
1975	1	1.6	5.2	1	1.1	5.2
1976	2	2.7	3.1	2	2.6	3.0
1977	3	4.6	1.6	2	5.0	1.1
1978	2	3.1	2.7	2	4.6	2.6
1979	1	1.6	4.6	1	1.3	5.0
1980	3	4.3	3.1	2	4.2	4.6
1981	2	4.2	1.6	2	2.0	1.3
1982	1	2.5	4.3	2	1.8	4.2
1983	2	1.7	4.2			

$C_P(t\text{-}2)$ and 0.13 for P_I. Observed and calculated chick production deviated on average by 0.59 these years (Fig. 2a). We used the following equation in the full model calculation:

$$C_P(t) = 5.7270 - 0.6319\ C_P(t\text{-}2) - 1.2564\ P_I(t) \quad (5).$$

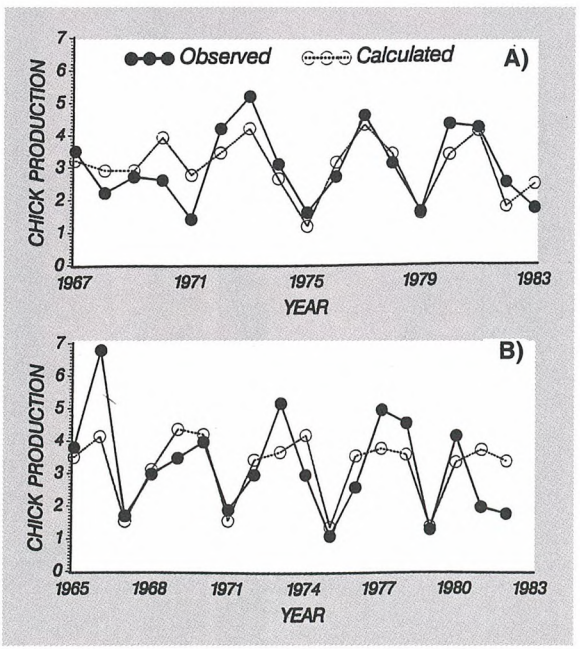

Figure 2. Observed and calculated chick production in: A) the Lövhögen (Sweden) inland test population during 1967-1983, and B) the Tranøy (Norway) island test population during 1965-1982.

Using equation 2, the regression analyses of data from Tranøy (see Table 2), for which the model included chick production two years earlier alone, explained only 12% of the multiannual fluctuation in chick production during 1965-1982 ($F = 2.10$, $df = 16$, $P = 0.17$). The model using the predation index alone explained 44% ($F = 12.50$, $df = 16$, $P = 0.003$), which was about the same as the full regression model (45%; $F = 6.12$, $df = 15$, $P = 0.01$). The P-value for each individual factor in the model was 0.60 for $C_P(t\text{-}2)$ and 0.009 for P_I. Observed and calculated chick production deviated on average by 0.87 (see Fig. 2b). The equation used in the full model calculation was:

$$C_P(t) = 5.8995 - 3.9063\ P_I(t) - 0.1178\ C_P(t\text{-}2) \quad (6).$$

Discussion

As most adult females in the spring were chicks two years earlier and as the number of chicks with four or more heterozygous genes increased significantly with increasing chick production, a significant positive association with chick production two years earlier for adult nesting females with four or more heterozygous genes was generated. Significantly more adult than juvenile females with four or more heterozygous genes suffered predation (Rørvik et al. 1999), in our study, therefore, chick production two years earlier was used as a measure of heterozygosity in adult nesting females. However, a significant association does not imply a causal relationship. It can also be a measure of the age structure of reproducing females. As older females might be better mothers (Hannon & Smith 1984), it could be argued that age alone, without genetics, affected the results. This is probably not the case, as chick production two years earlier ($C_P(t\text{-}2)$) was negatively related to chick production ($C_P(t)$ - equation 4), suggesting, if age related, that older females would be worse mothers. In addition, during the ptarmigan breeding season about the same extent of predation on juvenile as on adult nesting females was observed (Munkeby Smith 1994). It might also be argued that in fluctuating ptarmigan populations *per se*, there is a negative relationship between chick production a given year and two years earlier and that this alone, without genetics, generated the results. This is contradicted by the significant relationship between $C_P(t)$ and $C_P(t\text{-}2)$ in the Lövhögen test data only in the present study. In

Gåvålia and Tranøy, the regression model including chick production two years earlier alone explained only 19% and 12% of the annual changes in chick production, respectively. The same results in Gåvålia were observed when the percentage of females with high heterozygosity was substituted by the chick production two years earlier, even though there was no relationship between $C_P(t)$ and $C_P(t-2)$.

In the Gåvålia study area, neither females with high heterozygosity nor the proportion of females suffering egg predation alone could significantly explain observed annual changes in the chick production. However, when both the genetic and the predation components were included in the model (equation 3), a significant fit of calculated to observed chick production arose. A significant fit of calculated to observed chick production also was found when heterozygosity and percent females suffering egg predation were substituted with chick production two years earlier and small rodent density in the present year, respectively (equation 4). Hence, in both models the individual variables become significant only when included in the full model. In addition to a significant effect of the individual variables in the models, genetic variation of nesting females and predation and their substitutes improved the explanation of the observed variation in chick production if located as the last regression variable rather than the first variable. This may imply that predation and genetic variation of nesting females relates in a non-additive way when used to explain the fluctuation in the autumn number of juvenile willow ptarmigan in Gåvålia.

When using the same data set as Steen et al. (1988a) from Lövhögen (minus the first two years for starting of the calculations), the model explained 61% of the observed variations in chick production, compared with 25% for the curve-fitting model of Steen et al. (1988a). The better explanation found using the current model was mainly due to increased amplitudes of calculated annual fluctuations in chick production (see Fig. 2a). This is in agreement with several studies having shown the importance of predation in spring on the autumn number of juvenile willow ptarmigan in Scandinavia (Myrberget 1972, 1988, Marcström & Höglund 1980, Steen et al. 1988a) and that predation not only reduces the number of breeding birds, but does it in such a way that genetic variation among chicks at hatching becomes reduced, causing reduced viability and increased mortality among chicks (Rørvik et al. 1999). Hence, predation among nesting females enhances chick mortality, causing increased amplitudes of calculated annual fluctuations in chick production.

Generally, in the two inland populations (Gåvålia and Lövhögen) a significant effect of genetic variation of nesting females was found, generating a better fit between calculated and observed chick production when both predation and genetic components were included in the model than when predation alone was included. However, this was not the case on Tranøy. Here, the model explained about the same proportion of the annual variation as the model used by Steen et al. (1988a), and it did not improve the explanation (45%) compared with the model using predation alone (44%). The different results obtained between the two inland populations in Gåvålia and Lövhögen compared with the island population of Tranøy may have been caused by the different predator communities on a small island like Tranøy compared with inland locations. On Tranøy, predators settle when small-rodent numbers increase (Steen et al. 1988a). When small-rodent numbers crash, predator populations such as the weasel *Mustela erminea* are 'trapped' within the limit of the island. They must turn to alternative prey, mainly willow ptarmigan eggs and chicks. In addition to weasel, the main egg predator on Tranøy is the hooded crow *Corvus corone cornix*. In the inland locations, more complex predator communities are found, with important predators like the golden eagle *Aquila chrysaëtos*, gyrfalcon *Falco rusticolus*, red fox *Vulpes vulpes* and pine marten *Martes martes*. The mammalian predators are all important egg predators, and all these inland predators are important predators of breeding willow ptarmigan females (Munkeby Smith 1994). The simple predator community found on Tranøy might not cause any selective predation pressure on nesting willow ptarmigan, whereas this might be the case in the more complex predator communities found in the inland locations. For example, on Tranøy and other small islands, a large proportion of breeding willow ptarmigan females are located by pointing dogs while incubating (S. Myrberget, pers. comm.). In the inland study area, pointing dogs only occasionally flushed incubating females, except when these left their nests to forage (H.C. Pedersen, unpubl. data). With predators like the red fox and pine marten in the predator community, arrest of scent while incubating is probably a prerequisite for not being detected.

Acknowledgements - our sincere thanks go to J.E. Swenson and S. Omholt for constructive criticism of an earlier draft

of the manuscript, and to M. Øverland and two anonymous referees for comments on the present manuscript. The Norwegian Research Council of Science and Humanities supported the project.

References

Bergerud, A.T. 1970: Population dynamics of the willow ptarmigan Lagopus lagopus alleni L. in Newfoundland 1955 -1965. - Oikos 21: 299-325.

Chitty, D. 1967: The natural selection of self-regulatory behaviour in animal population. - Proceedings of the Ecological Society of Austria 2: 51-78.

Gyllensten, U. 1985: Temporal allozyme frequency changes in density fluctuating population of willow grouse (Lagopus L. lagopus). - Evolution 39: 115-121.

Falconer, D.S. 1981: Introduction to Quantitative Genetics. - Longman Group Limited, London & New York, 340 pp.

Hagen, Y. 1952: Rovfuglene og viltpleien. - Gyldendal, Oslo, Norway. 603 pp. (In Norwegian).

Hannon, S. 1988: Intrinsic mechanisms and population regulation in grouse - a critique. - Proceedings of the 9th International Ornithological Congress, pp. 2478-2489.

Hannon, S. & Smith, J.N.M. 1984: Factors influencing age-related reproductive success in the willow ptarmigan. - The Auk 101: 848-854.

Holt, E. 1953: Rypeproduksjon - værforhold. - Jeger og fisker, pp. 120-126. (In Norwegian).

Henderson, B. 1977: The genetics and demography of a high and low density of red grouse Lagopus l. scoticus. - Journal of Animal Ecology 46: 581-592.

Jenkins, D., Watson, A. & Miller, G.R. 1963: Population studies on red grouse Lagopus lagopus scoticus in north east Scotland. - Journal of Animal Ecology 32: 317-376.

Lance, A.N. 1978: Survival and recruitment success of individual young cook red grouse Lagopus l. scoticus tracked by radio-telemetry. - Ibis 120: 369-378.

Lindström, J. 1994: Tertraonid population studies, state of the art. - Annales Zoologici Fennici 31: 347-364.

Marcström, V. & Höglund, N.H. 1980: Factors affecting reproduction of willow grouse, Lagopus lagopus, in two highland areas of Sweden. - Swedish Wildlife Research 11: 285-314.

Moss, R. & Watson, A. 1991: Population cycles and kin selection in red grouse Lagopus lagopus scoticus. - Ibis 133 suppl. I: 113-120.

Munkeby Smith, E. 1994: Predasjon av reir og hekkende lirypehøner i rugeprioden. - Cand. Scient thesis. University of Trondheim, Norway. 40 pp. (In Norwegian).

Myrberget, S. 1972: Fluctuations in a North Norwegian population of willow grouse. - Proceedings of the XV International Ornithological Congress, the Hague 1970, pp. 107-120.

Myrberget, S. 1982: Fluctuations in microtine populations in an island area in northern Norway. - Fauna Norvegica Series A 3: 7-11.

Myrberget, S. 1985: Clutch predation on an island population of willow grouse, Lagopus lagopus. - Fauna Norvegica Series C, Cinclus 8: 82-87.

Myrberget, S. 1986: Annual variation in clutch size of a population of willow grouse, Lagopus lagopus. - Fauna Norvegica Series C., Cinclus 9: 74-81.

Myrberget, S. 1988: Demography of an island population of willow ptarmigan in Northern Norway. - In: Bergerud, A.T. & Gratson, M.W. (Eds.); Adaptive strategies and population ecology of northern grouse. University of Minnesota Press, Minneapolis, pp. 379-419.

Page, R.E. & Bergerud, A.T. 1984: A genetic explanation for ten-year cycles of grouse. - Oecologia (Berlin) 64: 54-60.

Pedersen, H.C. 1984: Territory size, mating status, and individual survival of males in a fluctuating population of willow ptarmigan. - Ornis Scandinavica 5: 197-203.

Pedersen, H.C., Steen, J.B. & Andersen, R. 1983: Social organisation and territorial behaviour in a willow ptarmigan population. - Ornis Scandinavica 14: 263-272.

Redfield, J. 1973: Genetics and selection at the Ng locus in blue grouse (Dendragapus obscurus). - Heredity 33: 69-78.

Rørvik, K-A. 1987: Electrophoretic separation of serum esterases in willow ptarmigan (Lagopus lagopus lagopus). - Hereditas 107: 191-195.

Rørvik, K-A. 1989: Inheritance of electrophoretic variants of serum esterases in willow ptarmigan (Lagopus lagopus lagopus). - Hereditas 110: 127-131.

Rørvik, K-A. & Steen, J.B. 1989: The genetic structure in Scandinavian willow ptarmigan (Lagopus lagopus lagopus) populations. - Hereditas 110: 139-144.

Rørvik, K-A., Pedersen, H.C. & Steen, J.B. 1990: Genetic variation and territoriality in willow ptarmigan (Lagopus lagopus lagopus). - Evolution 44(6): 1490-1497.

Rørvik, K-A., Pedersen, H.C. & Steen, J.B. 1999: An impact of genetic variation and predation on chick survival in willow ptarmigan Lagopus l. lagopus. - Wildlife Biology 5: 129-135.

SAS, 1989: SAS Institute Inc., SAS/STAT® Users Guide, version 6, Fourth edition, Volume 2. - Cary, NC, SAS Institute Inc., 846 pp.

Steen, J.B., Steen, H., Stenseth, N.C., Myrberget, S. & Marctröm, V. 1988a: Microtine density and weather as predictors of chick production in willow ptarmigan (Lagopus lagopus). - Oikos 51: 367-373.

Steen, J.B., Andersen, Ø., Sæbø, A., Pedersen, H.C. & Erikstad, K.E. 1988b: Viability of newly hatched chicks of willow ptarmigan Lagopus lagopus lagopus. - Ornis Scandinavica 19: 93-96

Steen, H. & Erikstad, K.E. 1996: Sensitivity of willow grouse Lagopus lagopus population dynamics to variations in demographic parameters. - Wildlife Biology 2: 27-35.

Watson, A. & Moss, R. 1979: Population cycles in the tetraonidae. - Ornis Fennica 56: 87-109

Watson, A., Moss, R., Parr, R., Mountford, M.D. & Rothery, P. 1994: Kin land ownership, differential aggression between kin and non-kin, and population fluctuation in red grouse. - Journal of Animal Ecology 63: 39-50.